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FOR THE STUDY OF THE CIRCULATION

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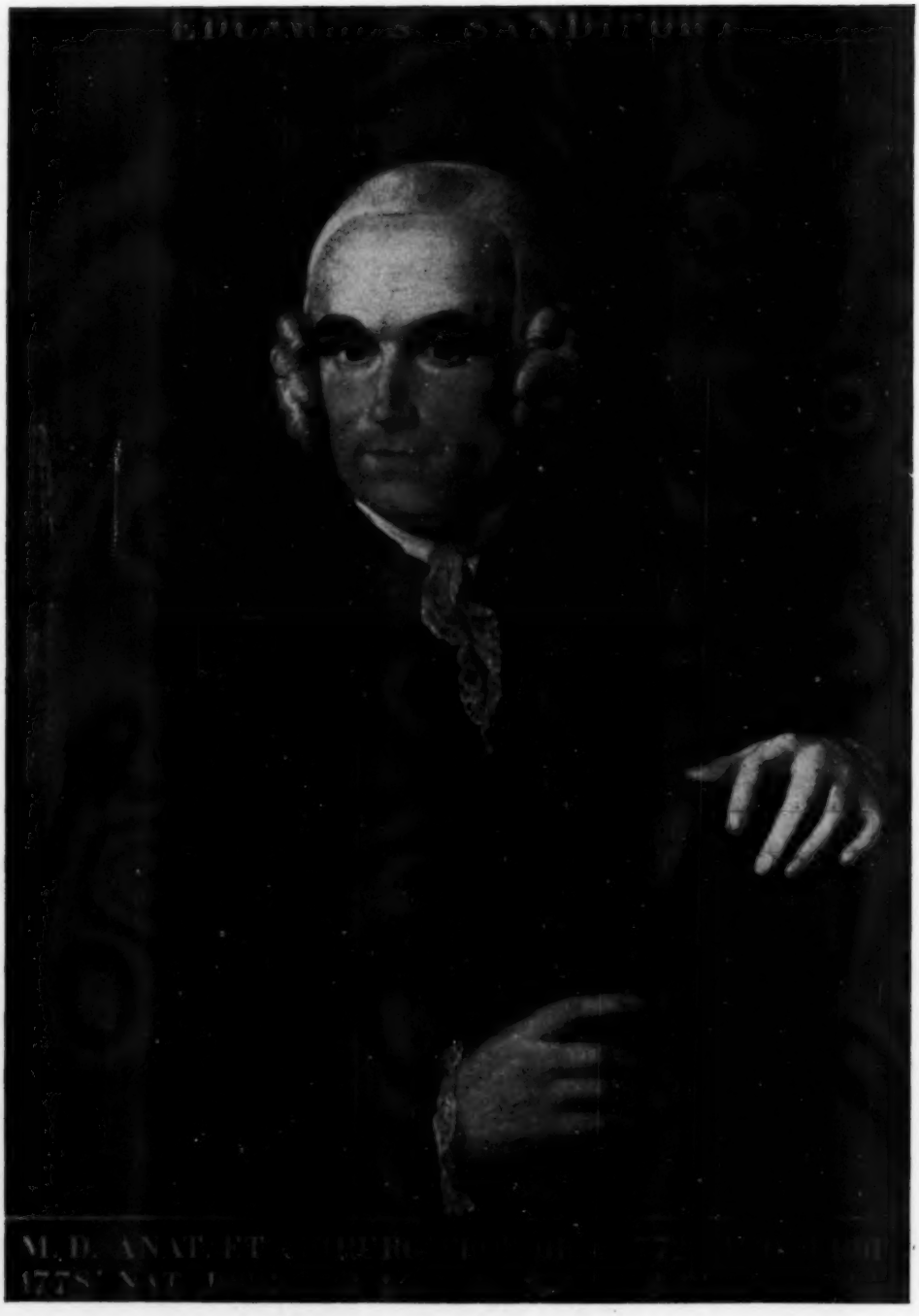

Eduard Sandifort (by an unknown artist)

The portrait was obtained through Professor H. A. Snellen, courtesy of J. Dankmeijer, Professor of Anatomy, University of Leyden.

American Heart Journal

VOL. 51 JANUARY, 1956 No. 1

FOREWORD

Some months ago I had occasion to look up the original description of the "tetralogy of Fallot" by Sandifort published in 1777, one hundred and eleven years before Fallot. It was so well done and so important historically that I was anxious to have it available for English-speaking readers who are not well acquainted with the original Latin text. Not knowing that the translation had been already done, I began to make my own laborious attempt. Happily, before I had gotten very far it occurred to me that there might already be somewhere an English translation. I, therefore, wrote to my friend, an authoritative medical historian, Ralph Major of Kansas City, to inquire. He replied that I would find a good translation of Sandifort's case report in the *Bulletin of the History of Medicine* by Lydian Russell Bennett. Thereupon, I went through the back numbers and came across the translation published in 1946. It was so well done that I was sure that it should become available to the medical public. Dr. Meakins, Editor of the AMERICAN HEART JOURNAL, agreed to accept this opportunity, and, with the permission of both author and publication, this excellent translation appears herewith.

Eduard Sandifort was born in Dortrecht, Holland, in 1742, and received his doctor's degree at Leyden in 1763. He became professor of anatomy and surgery in Leyden in 1772 and continued there until his death in 1814. Dr. Major writes of him as follows: "He was an outstanding anatomist, and, while he followed the methods of Albinus and was much interested in accurate and artistic illustrations, yet he treated anatomy in its relationship to disease and pictured diseased as well as healthy states. Cruveilhier called him 'the father of pathological iconography.' He wrote many books, among them *Observationes anatomico-pathologicae*, Leyden, 1777-1781, which contains the first known illustration of vegetative endocarditis in a patient with pulmonary stenosis and interventrical septal defect (Fallot's tetralogy)." He also prepared an *Atlas of Osteology* at the Museum in Leyden and was the first to describe anatomical and pathological details of the duodenum.

PAUL DUDLEY WHITE

SANDIFORT'S "OBSERVATIONES," CHAPTER I, CONCERNING A VERY RARE DISEASE OF THE HEART

LYDIAN RUSSELL BENNETT

CINCINNATI, OHIO

I

TETRALOGY OF FALLOT OR SANDIFORT?*

CURRENT newspaper and magazine articles frequently refer to heart trouble as the foremost enemy of health in America. Lowell Thomas, in his broadcast May 20, 1946, voiced a plea for the inception and support of a foundation to work out a solution for this problem. Added interest is developing due to the success of operations being performed at the Johns Hopkins hospital on children with congenital heart ailments, commonly known as "blue babies."

This condition has long been known as the Tetralogy of Fallot, which is characterized by four constantly present defects, pulmonary stenosis, dextroposition of the aorta, interventricular septal defect, and hypertrophy of the right ventricle. A Fallot (1850-1911) described this combination in *Marseille Médical* in 1888, and since then the disorder has borne his name.

The following translation from the Latin original of Chapter I of the *Observationes Anatomico-Pathologicae* leaves no doubt but that the disease now known as Tetralogy of Fallot is the same as the "very rare disease of the heart" described by Sandifort in 1777, more than a century before Fallot's publication. Hence this anatomical abnormality should properly be termed the Tetralogy of Sandifort.[1]

It is earnestly hoped that the translation of this section of Sandifort's work will be of interest to the medical profession, as the earliest description of this malady, and to Latinists generally, as an excellent example of early modern use of Latin for recording scientific data.

Reprinted through the courtesy of Miss Bennett and the Editor of the *Bulletin of the History of Medicine*. This reprint does not contain the whole of the article as published originally in the *Bulletin of the History of Medicine*, Vol. XX, No. 4, November, 1946. —Ed.

*"Tetralogy of Fallot or Sandifort?" I am informed by Professor H. A. Snellen that Sandifort published a second case of tetralogy, in much less detail, in Book 3, Chapter I, of his "Observationes."—Ed.

[1]"This combination of lesions was first reported by Sandifort." Brown, James W., M.D. *Congenital Heart Disease*, page 143. London, John Bale Medical Publications, Ltd., 1939.

Acknowledgment

Special acknowledgment is due Dr. Edward W. Miskall of East Liverpool, Ohio, whose suggestion regarding the importance of Sandifort's work prompted this research. Sincere thanks and appreciation are ascribed to the late Dr. James Stinchcomb, head of the Classics Department of the University of Pittsburgh, whose confidence in the ability of his pupils was a steady beacon on the often lonely road of scholarship, and to his no less able successor, Dr. Jotham Johnson. The invaluable aid and enthusiastic interest of Dr. Max Fisch and Dr. Dorothy Schullian, both of the Army Medical Library, are gratefully acknowledged.

II

EDWARD SANDIFORT*

Combining a scientific attitude far in advance of his time with a deep personal concern in the welfare of his patients, Edward Sandifort merits today both interest and admiration. He is justly celebrated as an anatomist, linguist, and teacher.

Born at Dortrecht, Holland, November 14, 1742, he received his preparatory training at The Hague, where his father, Gerard Sandifort, a teacher in the Reformed Parochial School, had moved: While studying the classical languages, Edward so far excelled his instructors in both Latin and Greek, not to mention his fellow students, that he was awarded an extraordinary prize, a thing not done heretofore.

At the age of sixteen he began his studies at the University of Leyden, where he came in contact with some of the famous anatomists of the day, such as F. B. Albinus, B. S. Albinus, Winter, Adriaan van Roijen, Gaubius, etc. He early showed a deep interest in anatomy and surgery, and pursued these studies diligently. On December 19, 1763, he was awarded the degree of Doctor of Medicine by one of his beloved teachers, Fredrik Bernhard Albinus. Sandifort's doctoral dissertation, written in Latin, was entitled *Dissertatio de Pelvi eiusque in partu dilatatione.*

He began his medical practice at The Hague, where he showed a continuous and ever increasing interest in natural sciences and the art of healing. At the age of twenty-three he began to publish his *Library of Natural Sciences and Therapeutics* (*Natuur-en Geneeskundige Bibliotheek*), four sections of which appeared every year for ten successive years. This magnum opus reveals Sandifort's linguistic ability, his voluminous foreign correspondence, his industrious nature, wide scholarship, and the importance he attached to the acquisition of a good library and the collection of all sorts of interesting items. The world is indebted to his *Bibliotheek* for the publication of many scientific articles, written by men of different nationalities, which otherwise would have been lost.

*Sandifort's first name is found in a variety of spellings, often abbreviated to *Ed.*, frequently *Eduard*, occasionally the Latin form *Eduardus* (as on the photograph), and the name is spelled with the anglicized form *Edward* in both the 1941 and 1947 editions of *A History of Medicine* by Arturo Castiglioni, M.D., translated by E. B. Krumbhaar, M.D., Ph.D., New York, Alfred A. Knopf, Inc.

Sandifort's growing medical practice was increased in 1769 when he was appointed City Physician. At the same time he took a deep interest in the inoculation of children with smallpox, and made a Dutch translation of the interesting English article on that subject, written by Thomas Dimsdale. His *Bibliotheek* contained articles on the same subject, written by his colleagues both at The Hague and Leyden.

Concurrently a serious cattle plague was raging in Holland. With others, Sandifort made a careful and serious investigation; then he wrote a treatise on his observations regarding the cattle cadavers, which was published in the *Transactions* of the Swedish Academy, and later translated into German. Sandifort was a member of the Swedish Academy and knew the language well. He translated the famous work of Rosen van Rosenstein, and greatly enhanced its importance with his notes and additions, so that the Dutch translation exceeded the original in value.

Several papers on anatomical pathology were published about this time. The treatise describing the autopsy performed in 1765 on a patient who died from a ruptured aorta, written in Latin, was deemed worthy of being included in the *Transactions* of the *Keizerlijke Leopold-Carolinische Akademie van Natuuronderzoekers*. One paper concerned the miscarriage of twins in which the one was fastened to the other by an appendage. Another paper gave a detailed anatomical study, with illustrations, of a deformed child, born with a split thorax and abdomen, so that the heart and viscera were protruding. Although Sandifort's writings appeared in rapid succession, his careful, painstaking methods of investigation show no sign of speed.

The confidence he inspired is evidenced by his rapid promotions. Appointed Lecturer in Anatomy and Surgery at the University of Leyden in 1770, he was promoted to Assistant Professor the following year, and to the rank of Professor of Surgery, Anatomy, and Medicine on February first, 1772. His inaugural address (in Latin) was published that same year.

Sandifort's students enthusiastically extolled his splendid teaching of anatomy, in which sphere he consistently excelled. After the death of F. B. Albinus in 1778, he took over the lectures in physiology and obstetrics. His orderly mind was shown in the organization and gradation of his lectures. A large number of cadavers was required annually, in order to give his students practical application along with theory. Some of his lectures on descriptive anatomy and on more general topics were open to the public. Perhaps it was due to these public lectures that Jacob van Noort and his wife, the father and mother of the "blue boy," not only gave their consent for the autopsy but even urged it for the benefit of posterity, as Sandifort tells us in the *Praefatio* to the *Observationes Anatomico-Pathologicae*, a thing which he declares is well worthy of praise and emulation, owing to the general feeling of horror and superstition regarding dissection in those days.

During his forty-three years at Leyden, as teacher and writer, Sandifort consistently followed the inspiring example of his teacher Bernhard Siegfried Albinus, whose methods he adopted, and whose scholarship and professional

skill he never ceased to laud, both in the class room and, what is more lasting, in his writings. In the *Praefatio* mentioned above he refers to him as Summus Albinus. In comparing the anatomical writings of these two men, a pronounced similarity may be noticed, both in the detailed methods of research and in the manner of presenting the material. In research both practiced frequent observation and painstaking examination of the minutest detail; in presenting the material they observed both exactness and clearness.

Yet there was no little difference, since Albinus treated anatomy as a natural science, while Sandifort believed the aim of the study of anatomy was to solve the problems of medical science, especially of internal diseases and surgical cases. Sandifort's lasting fame rests securely on his many contributions in the field of pathological anatomy. With the firm conviction that there is an inseparable connection between the material changes in affected organs, both regarding cause and effect, Sandifort urged post mortem examinations as sources of infinite value, especially since many pathological symptoms are either baffling or not obvious during the patient's life.

Another conviction of Sandifort's was that a careful record of clinical notes should be kept and published. In this he followed the idea of the great Boerhaave. Nowhere does Sandifort show the slightest tendency toward jealousy, selfishness, or egotism. As a youth he published in his *Bibliotheek* all his findings, with the hope that all Europe might be benefited. As a teacher he took obvious delight in the accomplishments of his pupils, and mentioned them by name in his articles, praising them for their careful work. He never hesitates to give full credit to any colleague from whom he received the slightest information.

But perhaps Sandifort's strongest conviction regarding pathological anatomy was that it should be represented graphically. To read a description in a book is not enough. If the knowledge gained through examination and observation is to be passed on advantageously to others, it must be clear to the eye as well as the mind. Hence Sandifort took great pains to produce the most exact illustrations to accompany his text and descriptive material. While some of his predecessors and contemporaries had illustrated articles with drawings, none approached Sandifort in either scientific precision or artistic skill. These exquisitely wrought illustrations were made either by Sandifort or under his direction. In this trend he was a forerunner of the modern exponents of visual education. While Albinus is justly celebrated as the reformer of descriptive anatomy, the great Frenchman Cruveilhier (1791-1874) rightly called Sandifort the "father of pathologic iconography."[2]

Edward Sandifort published sixteen books and pamphlets, ranging from articles of thirty-two pages to treatises of four volumes, mainly written in Latin, but several were written in Dutch or Swedish as mentioned previously. They all bear the stamp of wide scholarship and meticulous care. In addition to works already discussed, some of the most important include:

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[2]Hirsch, Dr. August. *Biographisches Lexikon der Hervorragenden Aerzte.* Vol. 5. Wien und Leipzig, Urban & Schwarzenberg, 1887.

Opuscula anatomica, 1784
Icones herniae inguinalis congenitae, 1781
Anatome infantis cerebro destituti, 1784
Observationes anatomico-pathologicae, 4 volumes, 1777-1781
Descriptio musculorum hominis, 1781
Exercitationes academicae, 2 volumes, 15 copperplates, 1783-85
Descriptio ossium hominis, 1785
Tabulae intestini duodeni, 1780
Notes to *Andreae Vesalii anatomici summi Tabulae Ossium Humanorum*, 1782
Museum anatomicum academiae Lugduno-Batavae descriptum, 4 volumes, 1793-1835

The last mentioned beautifully describes the contents of the famous Leyden Museum, and together with the *Observationes* gives magnificently engraved illustrations of congenital heart lesions and other viscera, herniae and intestinal obstructions, bony ankyloses and inflammations, ulcerative endocarditis, etc.

In addition to his scholastic, scientific, and literary activities, Sandifort also carried on an extensive practice in medicine. He was frequently called in consultation in difficult surgical and pathological cases, and his opinion merited the highest esteem. After the great naval battle of Doggersbank in 1781, the Town Council of Leyden assigned the establishment and direction of a hospital for the sick and wounded to Sandifort. Succeeding Fredrik Bernhard Albinus, he became president of the Collegium Chirurgicum, and later, when Adriaan van Roijen died, he was also made president of the Collegium Pharmaceuticum. In 1808 he was appointed surgeon and consultant at the court of King Louis.

Sandifort's varied activities, as well as his revealing writings, show his love for people and his keen interest in their sufferings. His sympathy for the "blue boy" pervades his account of the repeated attacks of pain and discomfort. His concern for the parents in their dauntless, but vain, efforts is mentioned several times. His professional and scientific interest is glorified by a gentle kindliness. After vast research and many autopsies, he referred to the wonderful structure of the human body as proof of divine wisdom.

In 1793 Part II of *Musei Anatomici Descriptio* appeared, but after that Sandifort published no major scientific work. Due to declining health, he requested some relief from his heavy academic duties. His son Gerard was appointed Prosector and Adjutor (1799), and two years later was made Professor extraordinary.

From 1805 Edward Sandifort devoted his attention entirely to Physiology, and he alone lectured on that subject. A salary increase of 400 florins was awarded him by the curators. When Holland was incorporated in the French Empire, and, due to this the University of Leyden was united with the French University, Sandifort retained his position as sole lecturer on Physiology. By translating foreign treatises and presenting current scientific discoveries to his pupils, Sandifort did his utmost to promote the study of physiology and the art of healing.

The latter years of his life were miserable because of poor health and the infirmities of age. Early in 1813 the head of the University granted his request to be relieved entirely of his duties. On February 12, 1814, his death occurred, a well-earned rest for one who had exclaimed so earnestly, "O how difficult it is to cure diseases within the breast! O how much more difficult to recognize them, and to give a sure diagnosis about them!" His entire life had been spent in directing the powers of a keen, methodical mind to the investigation and relief of human suffering. To the great University of Leyden, where he spent forty-three years, his untiring efforts contributed additional fame, both on account of the excellence of his teaching and the worth and extent of his writings.

III

The Observationes Anatomico-Pathologicae (1777-1781)

During the seventeenth and eighteenth centuries, Holland was the center of world book trade. More books were published there than in all the rest of Europe combined. Several printing companies flourished, vying with each other in hiring the best artists and engravers, and in producing the most beautiful books.

Sandifort's "*Observationes Anatomico-Pathologicae*" is in the Army Medical Library of the War Department, Cleveland Branch, where it was deposited for safe keeping during World War II. I have examined this original copy of the true book maker's art. It is a handsomely illustrated work on pathological anatomy, including, among other varied subjects, chapters on congenital abnormalities, herniae, bony ankyloses, ulcerative aortic endocarditis, renal calculi, etc. A. Delfos made the drawings, and R. Muys and Pieter de Mare engraved them. It was published by P. v. d. Eyk and D. Vygh of Leyden.

This large volume contains the four original books of the *Observationes*, with both text and notes written in Latin, and illustrated with 36 copperplates. Sandifort's lucid style, his linguistic scholarship, the orderliness and gradual development of the scientific material presented, and the pertinency of the content, today recommend this work both to the physician and to the Latin scholar. Those qualified to judge rate Sandifort's work almost equal with Morgagni.[3] No record of a previous translation into English of any part of the *Observationes* has been found by the writer.

The *Observationes* is dedicated to the curators of the University of Leyden and the members of the City Council of Leyden, "illustrious, generous, and very noble men." The Preface (Praefatio) contains six pages of Sandifort's own theories regarding the publication and free interchange of research among men of the medical profession. He also urges, in spite of the difficulty involved in making plates and diagrams, that figures are most useful in promoting and elucidating the study of anatomy and physiology. He had intended to use more illustrations than he did, but changed his plan in order to present the case of the "blue boy," described thoroughly in Caput I, as soon as possible, since he was convinced this was a rare case, and the information both valuable and useful.

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[3]Garrison and Morton. *A Medical Bibliography*. London, Grafton and Co., 1943.

Book I, published in 1777, contains 151 pages. The titles of the ten chapters are as follows:

I. De rarissimo cordis vitio.
II. De cordis, valvularum, arteriae aortae nonnullis morbis.
III. De hernia vesicae vaginali.
IV. De hernia intestino-vaginali, aliisque huius morbi speciebus.
V. De vasis emulgentibus, & pelvi renum.
VI. De calculo renali.
VII. De anchylosi inferioris maxillae.
VIII. De tumoribus utero annexis.
IX. De duro quodam corpusculo nervo auditorio adhaerente.
X. De appendice ex intestino ileo enata.

A detailed explanation of all the plates and figures and a complete index are placed at the back of Book I, along with definite directions to the publisher regarding the order and arrangement of the plates.

Book II (Liber II) of the *Observationes*, published in 1778, follows the same general plan of Book I. It contains 164 pages, with eight chapters and eight copperplates. A *Figurarum Explicatio* is also included. In the main, the subjects discussed pertain to obstetrics.

Book III (Liber III) was published in 1779. Following the arrangement described above, it contains ten chapters illustrated with ten engravings. The chapter headings refer to cases of hernia, kidney diseases, etc.

Book IV (Liber IV) appeared in 1781, discussing a wide variety of subjects in its ten chapters, illustrated with ten plates. It also contains some additional notes to subjects discussed in previous volumes, and a very comprehensive Index to all four volumes.

IV

Book I, Chapter I

Chapter I of Book I of the *Observationes*, whose translation is the subject of this present study, is entitled "Concerning a very rare disease of the heart." It runs from page 1 of the original publication to page 38, and includes three beautifully engraved plates, numbered I, II, and III.

The translation was made from photographic reproductions of microfilm copies of these pages, which were obtained through the courtesy of officials of the United States Army Medical Library, Washington, D.C., supplemented by a careful examination of the original copy, deposited at present in the Cleveland Branch of the Army Medical Library.

When the appropriate meaning of an unusual word or phrase was not immediately supplied by dictionaries, it usually resulted that a close examination of the context furnished an accurate and reliable significance.

I have kept page for page with the original printed text, for the convenience of the reader who may wish to study in greater detail the congenital malformation herein described.

The translation of Chapter I, entitled in Latin "De rarissimo cordis vitio," is presented on the following pages.

ED. SANDIFORT, OBSERVATIONS ON ANATOMICAL PATHOLOGY

Chapter One

Concerning a Very Unusual Malady of the Heart

Translated by Lydian Russell Bennett

Practitioners, with one accord, declare the diagnosis of many diseases to be very difficult, and they prove the observations of those who, by putting cadavers under the surgical knife, have revealed such causes and locations of diseases which not even the most skillful had been able to reach by conjecture; for so great is the difference, that although all diseases have been recognized and examined, as to causes and locations, we ought rather acknowledge with Boerhaave, that the character of the diseases, which afflict human bodies, is complicated and varied, having been observed now from many ages, and considered to have been described; but not as far as every variety of them could be exhausted, with the truly keen diligence of years of experience; whence indeed it results, that such new ones are always occurring, in recognizing which at the time no decision accurate enough for a doctor is made, in order that he may have and apply a mind correctly prepared by the precepts of his skill; indeed in truth doubt of the hidden cause, and uncertainty of the place affected, remain as obstacles to him who had won distinction in examining and curing other (maladies) (*a*).

If such great difficulty is met in recognizing most diseases afflicting different parts of the body, not less, certainly much greater (difficulty) presents itself regarding diseases of the chest; for concerning these we can speak with the highest authority, which Baglivus has professed regarding things which affect the lungs (*b*). O how difficult it is to cure diseases of the chest! O how much more difficult to diagnose them, and to give a sure prognosis about them! They even deceive the most experienced and the Leaders of Medicine themselves! And not infrequently they carry off the sturdiest men, breathing out health itself, in the midst of strenuous tasks, or in the gaiety of conversation, death being very sudden, or those to whom a certain illness seemed to threaten a rather remote death, they take from our midst suddenly, as if struck by lightning (*c*).

If the truth of this assertion should require proof, whether by the authorities or by direct observations, what a number of examples could be called to our aid, what an abundance of authors could be cited! Gross enlargements of the heart,

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(*a*) **Atrocius nec descripti prius morbi historia.** *Pamphlet*, **p. 98.**

(*b*) **Prax. Medic. Book I, chap. 9, p. 34.**

(*c*) **Mummsen de corde rupto, page 3. (About a ruptured heart).**

or of its auricles (*d*), erosions (*e*), ruptures (*f*); laceration of the vena cava (*g*); strange dilatation of the pulmonary vein (*h*); ossifications of the aorta and of the larger vessels (*i*), unusual stenoses (*k*), fatty tumors (*l*), erosions (*m*), aneurysmatic dilatations (*n*), and profusions of blood from these ruptured places into the cavities (of the chest) (*o*); degenerations of the valves (*p*); tumors hindering the action of the thoracic organs (*q*); rupture of the esophagus (*r*), all of which had produced various diseases in the living sick, the causes of which not even the most experienced doctors had been able to clear up by any skill, and show clearly the number of affectations to be remarkable, on account of which the action of either the lungs or heart, so necessary to life and health, is injured, disturbed, and impeded, the causes of which lie hidden, and would remain hidden forever, if dissection of the body were not begun (*s*). But indeed it is not necessary, either for these or other examples, that that wonderful con-

(*d*) O. Heurnius, Hist. & Observ. rar. Oper. Fernelii, annex to page 26. Pachioni Opera, pages 211, 214. Fantoni, Observ. Anat. Medic. 7, 19, 22, 29, 34. Meckel Hist. de l'Acad. de Berlin, 1750, art. 5. Morgagni de sed. & caus. morb. epist. 18, art. 4. Lancisius de subitaneis mortibus Oper. var. Tom. 1, pages 46, 48. De Haen rat. med., Vol. 1, page 251, Vol. 2, page 143, published at Leyden. Rat. Med. cont., Tom. 2, part 2, page 14, published at Vienna.

(*e*) Fantoni, Observ. Anat. Medic. 16, 22, 28. Blancardi, Anat. Pract. pages 94, 169, 202. Conf. cap. 2, not. (*f*).

(*f*) Ludwigius, Advers. Med. pract., Vol. 1, page 134, saw a ruptured right auricle of the heart. Petersen, Abhandl. der Schwed. Akad., Band 26, page 322, saw the same auricle gaping with a large perforation. This observation is also read in Geneesk. Verhandel. aan de Koninglyke Sweedsche Academie medegedeeld III. D. page 226. This is also called to our attention by a most beloved Brother, Polyatrus Haganus. Lemery detected a right ventricle ruptured through erosion, in the autopsy of the widow of the Duke of Brunswick undertaken by him, Mém. de l'Acad. des Sciences, 1732, page 428. The same ventricle (not the left, as Morgagni de sed. & caus. morb. epist. 27, art. 10, has it) split in the middle of the convex facies, Nichols found in George II, a most powerful King of Great Britain, Philosoph. Transact. Vol. 52, part 1, page 265. Uitgez. Verh. IX. D. page 352. The Bratislavian Doctors saw the same ruptured. Bresl. Samml. 1723, page 516. Hazon Journal de Médecine Tom. 9, page 516. The left ventricle ruptured by erosion was seen by Morand, Mém. de l'Acad. des Sciences, Year 1732, page 428, Harvey Exercit. de circulo sanguinis Tom. 1, page 149. Bohnius de renuntiatione vulnerum Sect. 1, page 63. Morgagni de sed. & caus. morb. epist. 26, n. 8, epist. 64, n. 15. Compare Mummsen de corde rupto.

(*g*) Lancisius Oper. Var. Tom. I, page 55.

(*h*) Hildanus Observ. Cent. 2, obs. 99.

(*i*) Vieussens traité sur la structure du coeur, page 109. Senac traité de la structure du coeur. Vol. 2, page 433. Lancisius de motu cordis & aneurysmatibus Oper. var. Tom. 2, page 250.

(*k*) Meckel Mém. de l'Acad. de Berlin, year 1750, art. 5. 1755, obs. 17.

(*l*) Stentzelius de steatomatibus, in principio arteriae aortae repertis, page 10.

(*m*) Act. Medic. Berol. Dec., 1, vol. 8, page 86. Morgagni de sed. & c. Epist. 26, art. 7.17. Epist. 27. art. 28. Weitbrecht Comment. Acad. Petrop. Tom. 4, page 263.

(*n*) Compare Verbruggen, formerly our very excellent pupil, now practicing medicine with the highest success at Delft, Dissertatio de Aneurysmate page 84, seq. where he dealt especially with aneurysms of the aorta, from a great many records, and at the same time he added a notable occurrence, illustrated with several figures. I myself described this disease both in Verhandeling over eene flagaderbreuk in de groote flagader, and in Nov. Act. Acad. Nat. Cur. Vol. 4, page 20.

(*o*) Morgagni de sed. etc. Epist. 17, art. 14, Epist. 26, art. 5, 15, 19, 21.

(*p*) Rather frequently Haller saw the tricuspid valves, thickened all over, to have grown stiff, and not controlled by their own muscles, or able to be driven on by the blood, which was emptied from the heart. Elem. Physiol. Tom. 1, page 333. Senac observed them hard, united by bony threads, joined to the walls of the heart. Traité de la structure du coeur. Tom. 2, page 433. Vieussens noticed the valves of the veiny left orifice of the heart (to be) bony. Traité sur la structure du coeur, page 102. Vieussens lib. cit. pages 107 and 109 mention those, which are found at the beginning of the aortal artery, (to be) unusual in bony hardness. Haller, lib. cit., Vol. 1, page 347. J. C. Themeln, Sammlung kleiner ungedruckter Ober-Ertzgebürgischer Schriften, 2, st. page 75. Meckel, Mém. de l'Acad. de Berlin 1755. Some things noted in the following chapter about the degeneration of the valves are worthy of comparison.

(*q*) Boerhaave, Opusc. page 111, seq.

(*r*) Idem lib. cit. page 98, seq.

(*s*) [A case report which was not pertinent to the present subject has been deleted.—Ed.]

formation of the heart should be described in this chapter, indeed this alone is sufficient to prove, not only that recognition of diseases of the heart is very difficult, but also the cure is often plainly impossible.

However, let no one believe that our work in describing this particular disease, or the work of others in outlining other plainly incurable conditions, is useless; for he is deemed worthy of equal praise, who by a definite plan proves the impossibility of the thing proposed, as he who unfolds what can be done through natural things. By this method of Boerhaave he fulfils his solemn promise (*t*), that notes should be taken as a constant rule, from which a doctor may be able to diagnose unknown diseases to be cured, and to distinguish cautiously from those which are manageable (and) admit the help of medical skill; thus he is guarded against being provoked often with harmful remedies, which it is impossible to make right; and such things ought to be put down (in writing), that I may say with Celsus, (*v*) not by what means any cure is effected, but that things may be recognized from their indications, and they would not think a doctor to have failed in his duty, if they should lose some (patient) thus. Indeed these things seemed to me of such importance, that I believed this particular case ought to be described in detail, especially when, in order that I might be able to perform this duty rightfully, the Parents, who by a rather unusual precedent, but worthy of praise and especially of imitation, suggested an autopsy themselves, the regular Doctor, a very learned man and Polyater Le Pla, and also Hahn, my very honored colleague, who was called in to consultation the last year, have informed me very courteously whatever things were observed from his birth clear up to his death, (but) I myself sought the true cause of death in the cadaver, along with the doctor just mentioned, and with three of my very earnest pupils, J.D. Le Pla, G.J. Le Pla, and van de Kasteelen, the first of whom prepared the figures very methodically for this special purpose.

Born of healthy parents, delivered in a healthy condition on Nov. 17, 1764, this infant was nursed not by his mother, but by his nurse, who, unsatisfactory because of convulsive symptoms, was not strong enough to nurse this boy, more than six weeks, afterward another one, who, being very healthy, cheerful, and overflowing with excellent milk, nursed him for a whole year, so that he grew very well, and just about completed the first year of his life in very good health, having been attacked by no disease, as far as either external appearance, or the unknowing habit of a baby to deceive, showed.

But when the first year of life had scarcely passed, the beginnings of terrible symptoms appeared, which afterwards sorely burdened the poor child. In fact, a bluish color of the fingers, even of the nails, not continuous, but now more and now less evident, attracted the attention of the parents, particularly when it could be ascribed to no tightness of clothing, which seemed blameable; however this (symptom) was not so evident that either the advice or help of a doctor was sought, since the infant seemed sound in other ways, and gradually he was beginning to support himself a little on his feet, and then to proceed slowly, so that he was walking about alone a little before his second birthday.

(*t*) Opuscul. page 112.
(*v*) Medic. Book 8, chapter 13.

Then in truth (the child) complained of extraordinary weariness, and soon his body would move as if scarcely able (to move) as much as children of that age are accustomed. Catarrh developed, attended by a heavy, troublesome cough, and as long as he stubbornly refused the medicine prescribed by a reputable physician, a great many spots, first red, then bluish, were discovered on his face the next day. The cough bothered him from time to time, movement was gradually more laboured, and when he had tired himself out, his face, hands, and feet would grow noticeably livid, indeed at that time the color of his lips and tongue often verged towards black, changing quietly again to the natural color; and these alternations were generally observed every day. In the meantime the height of the body increased greatly, the appetite was good enough, and there was no complaint except about fatigue, a pressing pain in the upper part of the head, discomfort, especially in the winter time, of such cold, even inside, that the hearth fire in the winter scarcely made his body warm, nor in the summer did the hottest rays of the sun exhibit the natural effect, much less could they produce perspiration.

At the beginning of March, 1767, a venesection was performed with this result, that both the discomfort and the pressing pain of the head were lessened for a time. The blood was then thick and dark, and after it had been chilled, the thickness did not separate itself from the serum.

Toward the end of the same year the child easily overcame smallpox, with no augmented distress at the time; after a few months he was sick with measles, then with false smallpox, and his main disease was neither increased nor lessened by these (illnesses). The symptoms mentioned, however, harassed his recovery from these diseases, and soon particularly the discomfort would agitate his body which then and often afterwards was accompanied by such violent palpitations of the heart, that they could be seen by the eyes, and indeed the heavy beats of the heart could be heard. Then riding was suggested in order that the body might be exercised without exhaustion, but it neither proved useful nor could be endured for a long time.

Gaubius, having been called into consultation in the year 1769, believed motion (activity), which could be made without exhaustion, to be absolutely necessary, and he advised that a cold bath should be given twice a day, in such a way that the water would reach to the knees the first day, to the abdomen the second day, and should reach higher on subsequent days, until it extended to the neck; and this (bath) having been administered for a quarter of an hour, motion (activity) should be started, but if this were impossible, the child ought to be placed on a bed, and its body ought to be rubbed briskly, however, he warned this remedy should be discontinued, if by chance the child should determinedly resist. Indeed such a bath has not often been given, for when once the chest had been immersed in the water, he was seized with such distress, that he utterly refused to enter the water quietly. And so with a gentle motion, venesection having been repeated from time to time, they tried to overcome the disease, or to make it more endurable, but without result. It remained in the same condition. A dry cough used to accompany the distress, when this had reached the highest degree, but was more tolerable, whenever the distress was

lessened. The exhalation of the mouth was unusual at the time, and should be compared with that which is accustomed to exude from an egg which has been freshly cooked and opened immediately.

This sad series of illnesses, now more, now less, bothered the child until the year 1774, then indeed his distress was so increased, the palpitations were so violent, that this attack far surpassed all preceding ones in degree; sinking spells, pressing pain in the head, swelling of the jugular veins, and either an agitation or a throbbing in them, have been observed. Since horseback riding was plainly impossible, he was driven in a carriage daily or every other day, in order that he might not refrain from all activity. A vein having been cut in the month of May, the distress was decreased again, and his body has been restored to rather brisk activity, so that immediately afterwards he walked around for an hour without great fatigue, and his parents revived their hope of restoring his health. But in the Autumn all the symptoms grew worse; his cough being dry and bothersome, bloody spittle, and presently pure blood, were spit up, for which reason blood was extracted from his body twice in the month of November, within the space of four days. However, the distress persisted, indeed immediately afterward it was so great that death seemed near, and the child himself felt that he would not survive long, very often declaring that the disease from which he was suffering, being entirely unknown to all, was incurable, and that no one was able to understand what he (himself) felt in the location of his heart. On the fourteenth of December, venesection was tried again and brought relief for a few days, but gradually the disease grew worse again, so that winter, very hard on the child himself, produced serious worries to the parents also.

In the following month of April, and also in July, venesections were repeated, relaxing and purgative potions were drunk in large quantities, and he survived this entire year, without feeling any relief. Fever was scarcely ever noticed through the entire course of the disease.

The things which Hahn prescribed early in the year 1776, when he was examining the child, brought relief for a time; however gradually the disease grew worse, activity was clearly impossible, and, if he exercised his body even a little, he used to drool an unusual amount of water from his mouth; he was seized with leipothymia, and for some time he was unable to see anything at all. Whatever methods were applied then were in vain, the distress was signally increased, especially when he went to bed; things which were pleasing before, no longer affected him at all, his face began to swell, his feet were troubled with oedema, he dragged out a very miserable life until March 8, 1777, when, seized with terrible suffering, he passed away.

I have mentioned very briefly the things which ought to be observed in the last year, since Hahn communicated with me very courteously the things he believed ought to be noted at that time, and which now follow in the words of my very honored colleague himself.

"I first saw the blue boy, about whom different rumors were going around the city, near the early part of 1776, having been called (in consultation) both by his father Jacob van Noort, a very excellent man and a highly respected citizen, and by his physician, the very distinguished Le Pla, who had charge of him from early infancy.

"I immediately recognized the disease, the renown and strangeness of which was lessened by the presence of the patient, as happens sometimes. For the boy was asthmatic, and after the slightest activity of the body he would breathe with such difficulty that his face and hands, as if in strangulation, would become livid, and immediately after would seem colored with a bluish pigment.

"But the cause of such extreme and long-lasting asthma was obscure; and it was not even consistent from the beginning of the disease. The parents asserted, the doctor confirmed it, that the boy was born healthy, that he did not show any indications of a badly affected heart during his first year; at length in the second year the blue color and signs of asthma appeared, the disease was not continuous, but now rather severe, now rather light, however such that, as the boy grew older, it grew worse along with his growing body.

"The appearance and figure of the boy was comely; his stature was tall for his age; the proportion of his limbs was right; there was always some difficulty in breathing, even after a rest, especially in the winter time; but far greater after bodily activity. The face was always somewhat swollen; the eyes were bulging and fixed, sure proof of some trouble; the color and appearance of his face in an acute attack of asthma were such as is accustomed to be seen in men who have been walking long and briskly against the winter wind. Then his cheeks, the end of his nose, his ears, and also his hands, fingers, and nails would grow livid; his lips, tongue, and the inside of his mouth were immediately afterward dark-purple. The pulsation of the carotid arteries was to be observed from a distance; the pulse of the arteries at the wrists was irregular and uncertain.

"The very changeable mood of this clever boy, generally difficult and somewhat quarrelsome, was suddenly gay for a short time, but still in such a way that the face and eyes of the smiling boy continued to show clearly the constant discomfort. Thereupon complaints of varied type used to return; of headache, especially along the sagittal suture, earache, pain in the breast and left side of the body, (of) nausea, pain in the abdomen extending sometimes to the pubic region, feeling of cold, and other annoyances.

"And reasonably, as different phenomena were noticed at different times, so the causes of the complaints were varied. But these things I saw repeatedly and consistently.

"1. Great difficulty of breathing after bodily activity, and noticeable pulsations of the veins in the neck.

"2. The face fuller than the proportion of flesh in the limbs warranted, and the same (the face) bluish between gasps for breath, the eyes distended, and sometimes suffused with blood.

"3. The urine always of full color, without sediment.

"4. Very slow digestion, not only when the appetite was poor, but also with increasing desire for food.

"5. Constant feeling of cold, although the skin was warm to the touch, which feeling of cold did not leave the boy, except in bed after a thorough warming of his body; for in the winter while sitting next to the hearth he used to complain about shivering and coldness, and also through the summer he used to seek the hottest rays of the sun or the kitchen fire.

"Finally, and I noticed this constantly, a soft (pliant) stomach, and also a discharge of blood from the nostrils, which, occurring at intervals, now and then brought great relief.

"The phenomena clearly showed that the descent of blood from the head and its passage through the lungs to be impeded; but what, what sort, and where the obstacle was, was not at all evident with any sure indications. The constant declaration of the parents and of my friend the doctor, that the disease was not congenital, increased the difficulty of the investigation. Because of this premise, the following conjecture seemed by no means absurd to me: the pernicious vapors of coal fires had injured the tender lungs in early infancy, and had made him susceptible to a stubborn asthma, since several times I have seen chronic diseases of the chest which were induced by smoldering coal fires which had been placed in a bedroom having no ventilation through the winter.

"I made many attempts, partly to reveal the cause and location of the disease, and partly to lessen the distress of the symptoms. The greater part of my efforts were useless, and neither did the remedies, except these three do any good.

"1. Leeches applied to the neck and behind the ears noticeably relieved the pains in the head.

"2. Several times when there was a heavily coated tongue, very foul breath, anorexia, and greater flatulence than usual around the diaphragm, a gentle emetic was obviously beneficial, and for several days.

"3. Cathartic pills moreover showed particular relief, if given with such moderation, that the excrement was kept soft, but not made liquid."

So much for Hahn.

Whoever studies a detailed history of a particular disease, whoever has learned the functions of the lungs and the heart and has observed the usefulness of those (organs), which sound Pathology has taught, what diseases ought, or are customary, to follow when either the lungs (*w*) or the heart (*x*), or at the same time the organs of respiration and circulation, do not perform their duties properly, will very readily place the root of all the symptoms observed in our boy in these disturbed vital functions. From these come the difficulty in breathing, the pain increasing with bodily activity, the violent palpitations, the fainting spells, the pounding (*y*) of the jugular veins; from these come the bluish color of the face, lips, tongue, and the dark-purple of the inside of the mouth; from these (come) headache, earache, pain in the heart and the left side of the body; from these the feeling of cold; and finally from these disturbances, which have appeared in the whole animal family, remaining illnesses are easily explained,

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(*w*) Ill. Gaubii Institut. Pathologiae Medicinalis, page 409.

(*x*) Id. lib. cit. page 418.

(*y*) Morand observed this agitation of the jugular veins, when polypus, present in the right auricle, penetrating even into the veins, was curtailing the free circulation of the blood. Mém. de l'Acad. des Sciences, 1732, page 432. Homberg saw the same indication in asthma, polypi were present in the aorta and in the pulmonary artery. Ibid. page 433. Compare Baader Observ. Medic. Incisionibus cadaverum illustr. obs. 1, page 8. Lancisius said this agitation was an unavoidable result, since the root of the vena cava, the ear, or the right ventricle, are dilated, because the blood, when the heart contracts and the opening of the right ventricle is not properly closed, is then driven into the vena cava. De motu cordis et aneurysmatibus Oper. var. Vol. 2, page 253.

and these things, which brought relief, (such as) bleeding from the nose, venesections, applications of leeches, prove the truth of such an explanation

But even if all these things could have been known while the patient was still alive, it would have been just as impossible to detect the true hidden cause, the barrier blocking respiration and circulation, since the more numerous the hindrances, so they are very different in nature and location, by which free circulation, free respiration is impeded; and except for relief measures (medical) skill has been able to advance not at all, indeed it could not advance, even if it had been possible to suspect and (or) recognize the true cause.

However, autopsies of those, whom similar maladies had previously bothered, had been able to represent as probable that the location and cause of the disease lay hidden in the heart, rather than in other parts of the body.

A baby, well formed, well nourished, (but) suffering from birth with difficult respiration, a weak, hoarse voice, leaden color of the entire body, cold extremities, lived only thirty hours; the lungs were strongly inflated, the blood vessels swollen with their own blood, the right ventricle of the heart, greatly expanded along with the pulmonary artery, but not even a trace of the foramen ovale could be discovered (*z*).

A young girl, who had been lying ill continually from birth, used to breathe in gasps, mainly on account of very feeble strength, and she had become ashen all over, as if from the color of her skin, at last she passed away, when she had almost reached her sixteenth year. Her heart had a small size and the point (apex) turned upside down. The left ventricle was formed like the right, and the right in turn like the left, and although wider because of this, the walls however were thicker. Likewise the whole right auricle was larger than twice the left, and fleshier than twice the left. Then also between each (auricle) the foramen ovale opened up just enough to admit the little finger. One of the three triangular valves had a normal size, the other two were rather small. The sigmoids however, which are placed in the opening of the pulmonary artery, indeed were near the base according to nature, but seemed cartilaginous in the upper part, having already even a very small piece of bone, and they were so entwined with each other in this part, that they scarcely left an opening, through which the blood could escape rather sluggishly. Moreover near that opening there were certain small fleshy-membranous prolongations, placed there with this idea, that they could supplement the alternations of the valves, by opening to the outgoing blood, but by checking its return (*a*).

Very difficult respiration, palpitation so strong that the ribs were pushed out of place, leipothymia, coldness of hands and feet, are read to have been observed in a man about twenty years old, in whose cadaver the heart was more swollen than usual, especially the right chamber, which was swollen with coagulated blood, the arteries and pulmonary vein were distended with coagulated gore, however the vein, entering the heart, was almost closed from within by a cartilaginous substance, so that it would admit only a thick needle (*b*).

(*z*) Vieussens Traité sur la structure du coeur, page 35, seq.
(*a*) Morgagni de sed. et caus. morb. Epist. 17, art. 12.
(*b*) Blancard Anat. Pract. pages 13, 14.

Difficult breathing, violent palpitations of the heart, leaden color of the lips, chilliness, weak, poor, uneven pulse bothered the pharmacist at Daffis; but after his death the valves in the opening of the left vein of the heart seemed to be so bony that they remained immovable, except for a small space for the blood entering the ventricle, whence, the free return of the blood from the lungs having been impeded, the blood from this organ could not be admitted, and the right (side of) the heart could not empty itself, but the whole (organ) was extended in an enormous way (*c*).

A man, twenty-five years old, used to have a bluish face, a weak, uneven pulse, and was bothered with strong palpitations, indeed so much so that he was not able to exercise his body by a rather strong motion without shortness of breath; pains in the heart increased, sinking spells were frequent, and generally extreme chilliness. The pericardium was found to be filled with a great deal of serum, which was clear, and without odor or taste; the heart was misshapen, its left ventricle was almost equal to two man-sized fists in size, and its substance had been stretched into a tender, fibrous sack; in its interior were hidden many polypi; its right ventricle was full of clotted blood, and the blood vessels of the lungs and also of the brain were swollen with a similar (kind of) blood (*d*).

Pains and palpitations were present in some (persons), in whom the aorta with its valves had ossified (*e*). A woman suffered for a long time with difficulty in breathing, and palpitation of the heart seized her, with the least extra motion. Her lungs were black and heavy; in the right ventricle of the heart a little clotted blood was found; the left (ventricle) was extended into a large membranous sac, in which there was a single polypus, which exceeded four ounces in weight; the arch of the aorta was completely bony, moreover the passage was so narrow that not even the little finger could be inserted (*f*).

This woman, who had been subject to pain, general trembling of the body, and palpitations of the heart from childhood (tender youth), had a very contracted aorta, which scarcely equaled half the diameter of the pulmonary artery, and her heart was grossly enlarged and flabby (*g*).

In all the above cases, more or less symptoms were present, which have been observed in our boy, in all (cases) the heart particularly suffered, but with the same defect in every (case). In our (particular) case the location and true cause of the disease also was in the heart, but the defect, very different from all the other (cases) described, has been deservedly said to be very rare.

The autopsy of the cadaver was begun with utmost care on March 10th. Only the heart with the neck (throat) was opened up, since symptoms had clearly proved the source and origin of the trouble to be hidden in this place (here).

Notwithstanding the color of the skin was not as bluish as in life, due to a very large supply of blood being stagnant in the heart and larger blood vessels; however the separation of these (blood vessels) and of the adjacent muscles

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(*c*) Vieussens lib. cit. page 103.

(*d*) Störck ann. med. sec. pages 153, 154, seq. published at Leyden.

(*e*) Deidier apud Vieussens, lib. cit. page 109.

(*f*) Störck lib. cit. page 171.

(*g*) Meckel Mém. de l'Acad. de Berlin. 1750. art. 5.

was tedious for so much blood was still present in the blood vessels, even in the smaller (ones), that it flowed continually, and was a very great hindrance, and so much time was spent, before the bones were entirely laid bare, and the thorax could be opened. Then however the sternum (breast bone), loosened from the clavicle, the muscles separated, six upper ribs broken in pieces on each side, (the sternum) has been bent back toward the abdomen.

Thus the interior of the thorax was opened up, (but) the pericardium did not appear (*h*), as is customary, as if encircled and included by the lungs, but only one mass was seen, almost completely filling the entire cavity, and compressing the lungs as much as possible. However, this mass was the pericardium, holding the heart, greatly distended and very full of blood. It was stretched out from the diaphragm (which extended to the fifth rib on the right side, but to the sixth (rib) on the left) to a place midway between the first and second rib, and so filled the lateral parts of the thorax, that only the anterior part of the right lung could be seen, namely, the edge of the upper and middle lobe, but of the left (lung) only a very small part was showing from the side and upper part. Above the pericardium the superior vena cava was evident, along with the beginnings of the subclaviae, still covered with part of the thymos; it (vena cava superior) was swollen with black blood.

When the pericardium was dissected, a certain amount of water flowed out, not so much indeed, but that a far greater (amount) of such is often given off in cadavers, not affected with dropsy.

However the heart, separated from its sac, was very swollen, but not evenly, both ventricles were not distended the same amount, but the right ventricle, along with the sinus and auricle next to it, was extremely swollen with blood, (so that) it was much larger than the left, and all the veins running along the external surface of the heart, (the branches of the coronaries), were so dilated almost to the finest ends, that even the most successful injection could not possibly have made them more distinct.

The veins springing out from under the subclavian muscle, especially the jugular, were grossly enlarged with blood, which was thin and black. The superior cava, where it extends within the pericardium, was not much larger than its natural (state) in capacity, but the inferior (cava) was expanded much more than usual. The pulmonary veins also were distended, but not beyond moderation. The aorta was dilated larger than customary at the beginning, but the pulmonary artery was so greatly contracted in this section, where it emerges out of the heart, almost to a two-pronged fork shape, that it drew the attention of all to it. No trace was evident of the ductus arteriosus, or rather at this age, of the ligament, extending from the pulmonary artery to the aorta, once a passageway.

The lungs from the outside manifested no imperfection, however, being small, compressed, and not very extensile, they showed (plainly) enough they had not been sufficiently strong to perform their function properly, in every sense.

(*h*) Compare Eustachii Tab. 9, f. h. i. k. f. k. in the right side of the thorax, f. k. h. i. f. k. in the left side, x. x. x. x. Then in this capacity the pericardium n.w. enclosed as if by the right lung n.o.p. and by the left q.r.

But the external examination of the heart showed that the location of the trouble, and the only source of all the symptoms, was concealed in itself, (the heart). And so, after all the blood vessels had been tied, both those which it receives from the trunk or sends to it, and also those by which means it connects with the lungs, this (heart), separated from the trunk and from the organs of respiration, was subjected to further examination.

Plate I (Tab. I)

At first the right (*i*) sinus was opened with the auricle (*k*), in order that, after an ample amount of the thin, black blood had been let out, this cubic extent could be examined first. In the wall lying between the right and left sinus the foramen ovale showed an opening (*l*), which could admit a rather thick pencil.

(*i*) Tab. I. d.
(*k*) Tab. I. e.
(*l*) Tab. I. i.

A finger, having been put into the right ventricle, then curved around in the apex, and having moved forward toward the orificium arteriosum (*m*) of the pulmonary artery, which usually springs out from this ventricle, could detect that orifice in no way, but was admitted without any difficulty into the other

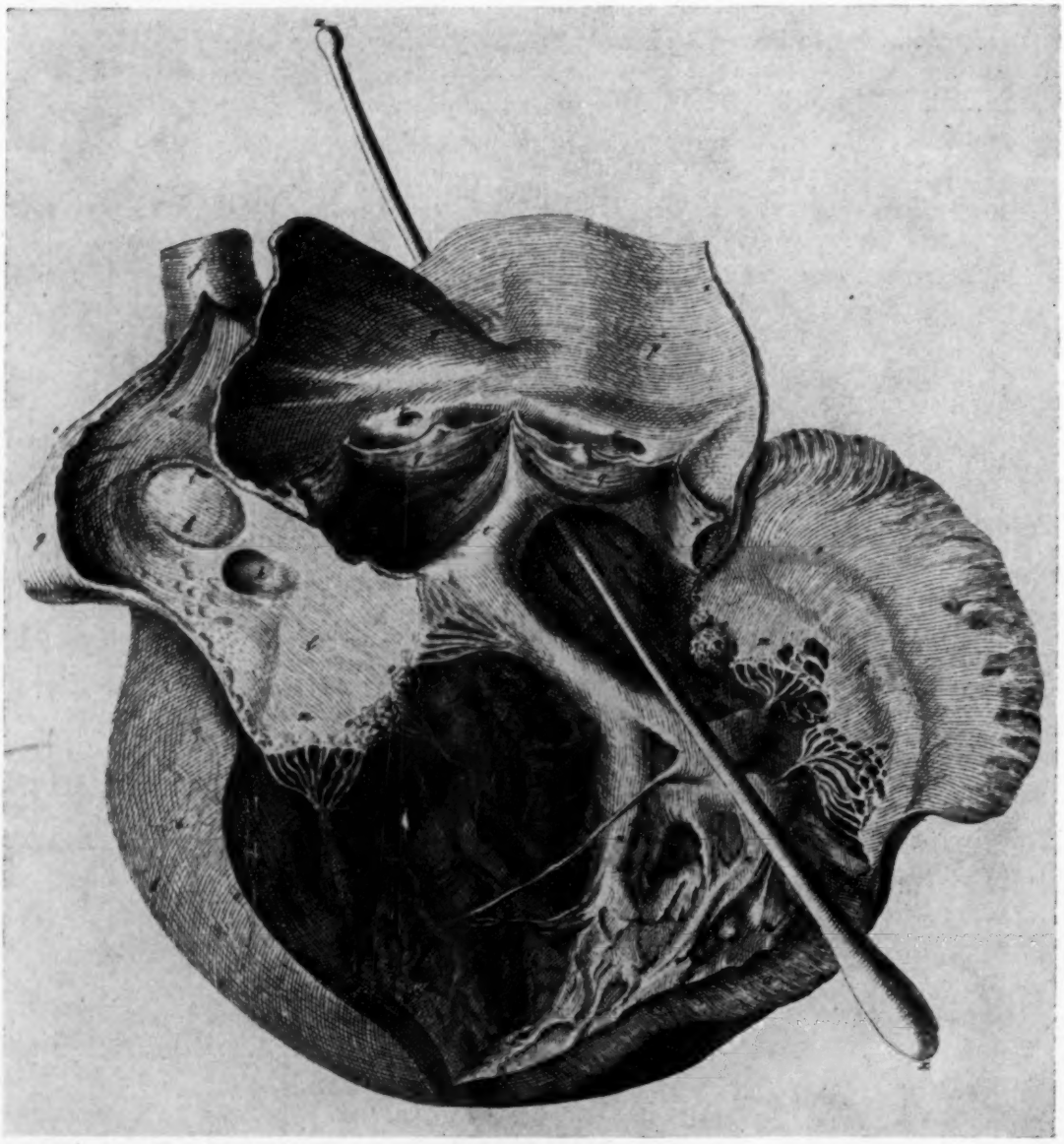

Plate II (Tab. II)

one, which was very capacious. But how great was the surprise of the onlookers, how great equally was my own surprise, when we saw the point of the finger to stretch into the aorta (*n*), which is not at all accustomed to maintain communication with the right ventricle, in conformity with otherwise constant laws of nature!

(*m*) Tab. I. P.
(*n*) Tab. I. q.

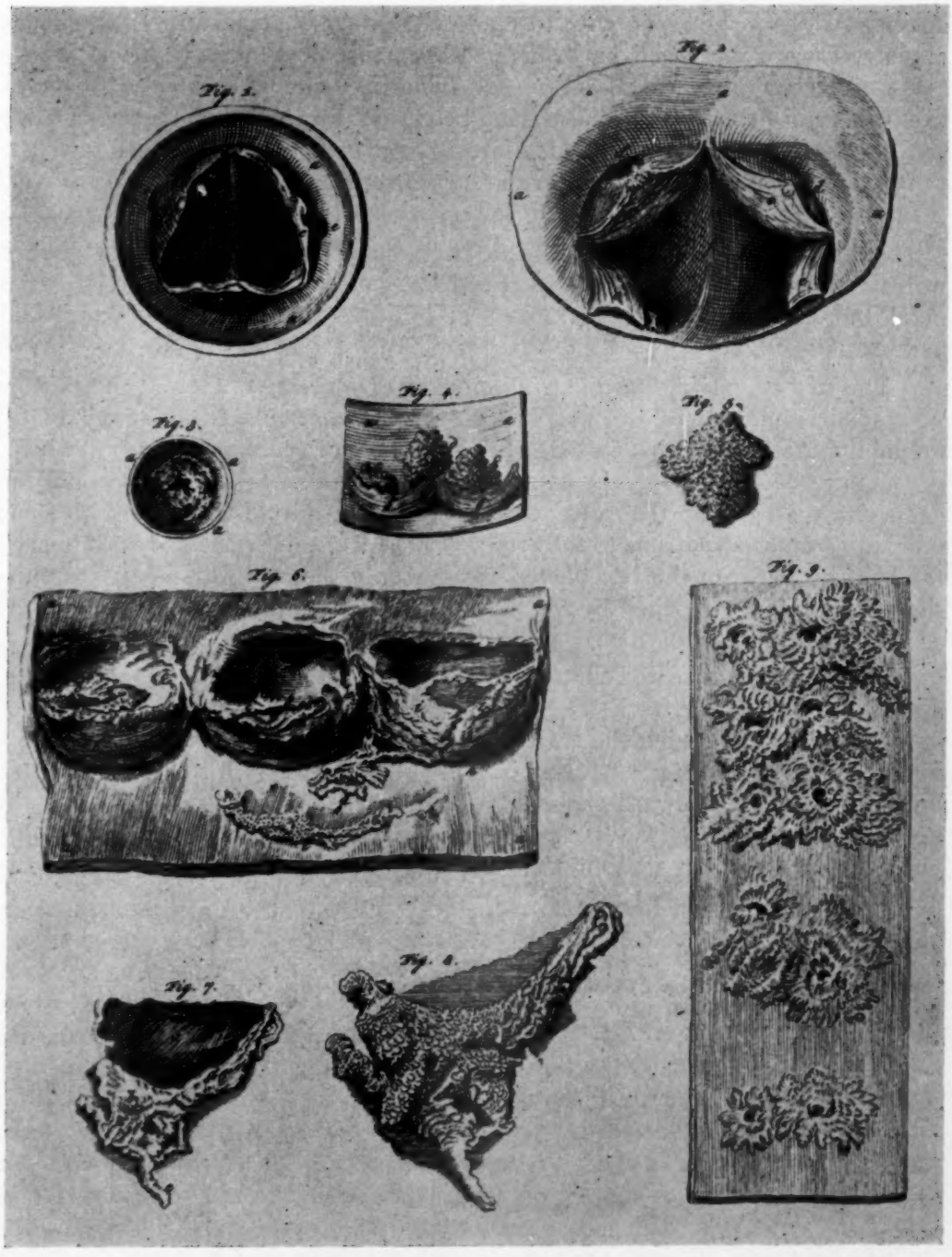

Plate III (Tab. III)

On account of this, the right ventricle in the part opposite that valve, behind which is the entrance to the orificium arteriosum, was split (*o*) all the way to the apex, and then not only the finger, but also the eye, when the aforementioned valve (*p*) was pushed aside for a while, could detect the capacious

(*o*) Tab. I. a. a.
(*p*) Tab. I. pone p. (back of p).

opening of the aorta, along with its smooth edge (*q*), beyond which the finger, moving forward and then backward, reached into the other ventricle of the heart, the left. The arterial aorta (*r*) having been cut, at a sufficient distance from the semi-lunar valves, and having been examined, showed that same edge (*s*), by which the orifice was divided into a larger part, connecting with the right ventricle (*t*), and into a smaller part, ascribed to the left ventricle (*v*).

And thus the arterial aorta was springing from both ventricles, and had to receive all the blood from both. This beginning of the great artery was very worthy of notice, but, another section having been made, it could be seen much more clearly by the eyes; after the orifice of the pulmonary artery had been examined first.

This artery, having been cut off above the valves (*w*), showed these to be very small, almost rigid (*x*), and blocked (*y*) by a certain granular substance, resembling fleshy growths, so that only a very small space was left (*z*), which, permitting the entrance of the point of a rather thin stylus, proved (to be) the passageway for this to the right ventricle, and transmitted the same with some difficulty from the ventricle to the artery (*a*). After this orifice had been opened (*b*) halfway between the two valves, we saw only the two pairs of valves, misshapen, and partly blocked by the aforementioned granular substance (*c*), just as it appeared (*d*) in the artery above the valves.

Then, having dissected that part of the right sinus, which is near the aorta, along with the artery itself (*e*), in the very opening appeared two untouched valves (*f*), back of which the entrances (*g*) of the coronary arteries are placed, a third valve had been cut halfway in two (*h*). The smooth edge, rounded (*i*), separating the way toward the right (*k*) and left ventricle (*l*), closing on the upper side the wall halfway between the two ventricles, showed up beautifully, and left no doubt at all, but that the large artery (*m*) would communicate equally with the right (*n*) and left (*o*) ventricle.

(*q*) This margin, afterward shown more clearly in Tab. II.t.
(*r*) Tab. III. Fig. 1.2.a.a.a.
(*s*) Tab. III. Fig. 1.2.f.
(*t*) Tab. III. Fig. 1.2.h.
(*v*) Tab. III. Fig. 1.2.g.
(*w*) Tab. III. Fig. 3.a.a.a. Fig. 4. a.a.
(*x*) Tab. III. Fig. 4.b.b.
(*y*) Tab. III. Fig. 3.b.b.
(*z*) Tab. III. Fig. 3.c.
(*a*) Tab. II. x.x.
(*b*) Tab. III. Fig. 4.
(*c*) Tab. III. Fig. 4. b.b.c.c.
(*d*) Tab. III. Fig. 4.d.
(*e*) Tab. I. p.d.q.
(*f*) Tab. II. q.q.
(*g*) Tab. II. r.r.
(*h*) Tab. II. s.s.
(*i*) Tab. II. t.
(*k*) Tab. II. w.
(*l*) Tab. II. v.
(*m*) Tab. II. p.p. Tab. III. Fig. 1.2.a.a.a.
(*n*) Tab. II. w. Tab. III. Fig. 1.2.h.
(*o*) Tab. II. v. Tab. III. Fig. 1.2.g.

The small left sinus, having been opened up, showed nothing except the opening of the foramen ovale. The left ventricle also displayed nothing unusual, except the margin in the first part of the aorta which has been mentioned. In the thickness of the ventricles not as much difference was noticed as it is customary for this place to have, but the right, if not thicker, had the same thickness as the left. Nowhere was polypous matter observed.

As long as there was such a great defect in the primary organ of circulation, which proved a very true source of all the trouble, we left the remaining cavities of the body intact, since in fact these had exhibited no disease at all, which could not be explained by an experienced person from the impeded circulation of the blood. For either I am greatly mistaken, and all those, who stood around me while I was dissecting, or those who heard me explaining the case, are mistaken, or (else) all the symptoms are caused easily by this one defect of the heart. They who recall to their minds the natural construction and work of the heart, who compare the same with the degeneration just described, understand well what a change took place in the circulation of the blood, what a change in the function of the lungs.

Perhaps some one will ask, should this defect be said to have been acquired, or was it really congenital? That it did not exist from birth, could perhaps be believed, because in the first year (of life) it gave no signs of its presence; free, even respiration was seen, in a word, a very healthy child lived. But then other questions arise; for what reason was the wall between the ventricles torn? or why was the margin with blood flowing near and past it continually, so greatly reduced in weight, that it seemed to have been that way from the very beginning of infancy? or why the aorta, with continuous expansion, certainly receiving almost all the blood from the whole heart, so changed regarding location, that now it rises from both ventricles, while before it extended to the left only? That the pernicious fumes of burning coal had injured the tender lung in early infancy, and had caused susceptibility to chronic asthma, was the conjecture of the very famous Hahn, with the parents and the Doctor repeatedly declaring, that the disease was not existing at birth. How much such fumes may injure the lungs, how often they may be fatal, is very important, and since the lungs could not be passed through, for some cause or other, it is evident from observations, that the blood had rent asunder either the pulmonary artery (*p*) or the valves (*q*), or to have distended the right ventricle as much as possible (*r*), or this same (ventricle) to have been suddenly ruptured (*s*). However, it is scarcely credible that a child could live eleven years, if from this cause such a laceration of the middle septum had been concealed between the ventricles. However the aberration could be ascribed to the aforementioned cause, if the beginning of the pulmonary artery, which was very constricted, had been, in the contrary, of unusual size, and the ductus arteriosus, to be found in a foetus between the

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(*p*) Matani de Aneurysmaticis praecordiorum morbis. Edit. Francof. page 145.

(*q*) Cowper Myotomia reformata Tab. 38. Fig. 3.a.a.a. Bassii Observat. Anat. chir. Medic. Dec. 1. pages 113, 114. Tab. I. Fig. 2.h.h.h.h. Miscellanea Acad. Nat. Cur. Cent. 1. page 171.

(*r*) Mém. de l'Acad. des Sciences, 1730, page 158. Vieussens Traité sur la structure du coeur. page 102. Compare note (*d*), page [10].

(*s*) See note (*f*), page [10].

pulmonary artery and the aorta, diverting blood from the pulmonary artery into the aorta had been greatly dilated (after a few months this is generally closed, however in the second year, even in the third, and indeed in an adult man, it has been seen open (*t*), but not even a trace of it (ductus arteriosus) has been uncovered in this body); for then it could be concluded, that the blood, driven forward by the great strength of the heart, but not admitted by the lungs, had again entered and enlarged the path, which it had deserted from birth. And truly it is permissible (*v*) to conclude from experiments that, in this case, so many diseases would have sprung up, that the child would hardly be strong enough to survive, for it is not possible to deprive a human being of the free use of his lungs, after these have performed their function for a considerable time, even if the circulation may be vigorous through the rest of the body, with these partially intact. (*v*)

That the disease was congenital seemed to me the far truer conjecture; nor does it contradict the fact that the child led a very healthy life in his first year of age, for at this time, since the lungs were not performing (*w*) their functions in full because of scarcely any motion and the very quiet life of the child, the defect, which actually was already present, was able to remain hidden, until the infant should exercise his body in (some) activity. But I am much more emboldened in this opinion of mine, since, although I have called the defect very rare with the highest authority, it is not, however, without (previous) example, (such as) the right ventricle communicating both with the aorta and with the pulmonary artery; in truth, the aorta being required to receive blood equally from the right and left ventricle.

For in an embryo, bordering on a monstrosity, which was dissected at Paris, Stenosis saw, beside other parts degenerating from the natural state, the heart, the liver, ventricle, spleen, almost all the intestines, and even the right kidney left protected by no covering, and the cavities of the chest and abdomen open. Before other things, however, the peculiar formation of the arteries, projecting from the heart, deserved his attention and astonishment. Something new in the front part seemed to indicate the artery of the lungs, much more arched than the aorta; therefore he opened this from the right ventricle of the heart all the way to the very substance of each lung, at the same time he clearly recognized that the duct, which leads from the artery of the lungs into the aorta, conspicuous elsewhere in every foetus, was entirely lacking here. But when he had opened up the right ventricle, a stylus pushed upwards next to the septum, found an opening extending even into the aorta, (and) with equal ease it next moved up from the left ventricle into the same aorta. Here there were three openings of the right ventricle, one from the auricle, two into the arteries, and this same duct of the aorta, common to each ventricle, formed (*x*) a double orifice in the middle septum of the heart.

(*t*) Conf. Halleri Elem. Physiol. Vol. 3, pages 161, 162. Vol. 8, part. 2, pages 9, 10.

(*v*) Haller lib. cit. Tom. 3. pages 314, 315.

(*w*) The Parents have rather frequently affirmed that, in the first year, the baby scarcely ever cried out, he did not cough, and, according to their word, he lived a very peaceful life. However, it is evident that crying would set in motion the dependent lung, fill his blood vessels with blood, and speed up the circulation through the inward parts. Haller Elem. Physiol. Tom. 8, part 2, page 10.

(*x*) T. Bartholini Acta Hafniensia, Tom. 1, n. 110, page 200.

Finally, what the Doctor ought to do, if he should come in contact with a patient with the same symptoms, which we have mentioned above, is easily concluded from the foregoing. Free circulation and free respiration have been checked, therefore it is well to apply as relief anything which, by decreasing the blood supply, makes its circulation easier, such as venesection and the application of leeches; anything which removes the thickness of the blood, and makes its first trips pure and free. Cathartics therefore will give the best success; foods easily digested, from which the best chyle is prepared, will satisfy the same purpose; very light activity should be permitted, but everything should be avoided, which is liable to tire the body or speed up the circulation of the blood very much. In our case, gentle emetics brought relief, so much indeed that at times the child would ask for such medicine, according to the assertions of the Parents, and also the observation of Hahn, at whose suggestion these (emetics) were taken; but these do not seem praiseworthy in other cases; for there is a greater congestion of blood in the head than necessary, the blood vessels are distended much too full, the return of the blood from the head is impeded; indeed in the very act of vomiting, even in those persons, who do not have an excessive amount of blood, the face is red and swollen, the head aches, the eyes, suffused with blood, water, spots are seen in front of the eyes, dizziness occurs, because the supply and force of the blood are diverted toward the head, and respiration is hindered by vomiting (*y*), therefore, physicians warn that emetics should scarcely ever be advised for patients suffering with plethoric, asthmatic, or like ailments.

1385 Burdette Ave., Apt. 14
Cincinnati 6, Ohio

(*y*) Conf. Van Swieten Comment. in Aphor. Pract. Boerhavii Tom. 3, page 266.

Original Communications

COMPARISON OF TWO SYSTEMS OF VECTORCARDIOGRAPHY WITH AN ELECTRODE TO THE FRONTAL AND DORSAL SIDES OF THE TRUNK, RESPECTIVELY

H. C. BURGER, D.Sc., J. B. VAN MILAAN, D.Sc., AND W. KLIP, M.D.

UTRECHT, NETHERLANDS

THE SYSTEM OF THE EQUILATERAL TETRAHEDRON (W_4)

THE application of vectorcardiography is hampered by a great number of systems that are being used side by side. The number of systems in daily use is not much smaller than the number of cardiologists who occupy themselves with vectorcardiography. It does not appear likely that in the near future a radical standardization will be brought about whereby one system will be maintained unanimously. A comparison of different systems is therefore necessary. This is not the first time we have undertaken such a study. We have investigated already three systems, viz: the "tetrahedron" system proposed by Wilson and associates,[6] which we called W_4, and the two systems suggested by us, B_1 and R_2 (Burger, van Milaan, and den Boer[5]). It then appeared that the agreement of W_4 and B_1 was better than of W_4 and R_2.

Since W_4 is very often used while, on the other hand, B_1 has the advantage of a physical foundation, we are of the opinion that a further comparison of W_4 and B_1 will be of importance. In doing so we have not confined ourselves to a simple comparison. We have tried, while maintaining the place of the electrodes of W_4, to improve the agreement of the vectorcardiograms according to both systems.

The relation between the three components X, Y, Z of the heart vector and the potential differences LR, FR, and WR between the four electrodes L, R, F, W of the system W_4 is given by the equation stated earlier:

$$\begin{aligned} &\text{(lateral)} && X = 40\ LR \\ &\text{(sagittal)} && Y = 16\ LR - 49\ WR + 16\ FR \\ &\text{(vertical)} && Z = -23\ LR + 46\ FR^{*} \end{aligned} \qquad (1)$$

The ratio of the coefficients follows, by means of a simple calculation, from the properties of the regular tetrahedron. It has been remarked earlier that

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*In the cited article (Burger, van Milaan, and den Boer[5]) Z and Y in these equations (p. 403) have been interchanged.

Wilson and associates did not state the absolute quantity of the coefficients We have chosen it in such a way that the agreement with B_1 was as good as possible.

This chain of thought can further be continued by changing all coefficients of either of the systems W_4 or B_1 so that the agreement is further improved, in which case we have the choice of changing either W_4 or B_1. We decided on the first for two reasons, namely (1) B_1 is physically founded; (2) the horizontal and frontal projections of W_4 are often so small in the lateral direction (X) that particulars are difficult to estimate by it, especially so in the horizontal projection.

The changes of the coefficients may thoroughly change the aspect of the loop, but this is not very real *mathematically*. It is a so-called linear transformation, that is, a change of a well-defined simple type. Particular cases of linear transformation are rotation, one-sided enlargement, and similar enlargement or reduction. Every conspicuous local peculiarity like a nick or a notch remains in similar transformation. The form of the loop may thereby change, so that from stretched or elongated it may become about circular. Such a transformation is extremely important for the comparison of vectorcardiograms in practice.

DETERMINATION OF THE PLACE OF AN ELECTRODE IN THE IMAGE SPACE

As is known in the system of Wilson and associates (W_4), the image points (Burger and van Milaan[4]) of the electrodes L, R, F, W in the image space are to be found in the angular points of a regular tetrahedron, of which the plane LRF is vertical and the edge LR is horizontal. When adapting this to B_1 the points LRF are to be found in the angular points of a scalene-angled triangle not lying in a vertical plane. This triangle is determined by the coefficients pertaining to the system B_1. So there remains only the problem of determining the place of the back electrode W in the image space in which the triangle LRF is situated. This can be done by observations on subjects. Because of the differences in anatomy and electrical heterogeneity of the trunk of different individuals, these investigations must be carried out on a sufficiently large number of subjects.

For this purpose we first applied a method proposed by Becking.[2] It is based on the following simple but only mathematically representable reasoning.

The potential difference W R of the back electrode W and the right arm R, chosen as the zero electrode, is a linear function of the components X, Y, Z of the heart vector:

$$WR = aX + bY + cZ \text{ (Burger and van Milaan}^{2}\text{)} \qquad (2)$$

In this formula WR, X, Y, Z, are functions of time and a, b, c are constant. These constant coefficients are the coordinates of the desired point W in the image space (Burger and van Milaan[4]). In order to determine a, b, c we express

the components X, Y, Z of the heart vector in potential differences LR, FR, BR, which play a part in the system B_1. We found in relative measure:

$$B_1 \quad \begin{cases} X = 54\ LR + 8\ BR + 16\ FR \\ Y = -12\ LR + 40\ BR + 46\ FR \\ Z = -10\ LR - 6\ BR + 26\ FR \end{cases} \qquad (3)$$

(Burger, van Milaan, and den Boer[5])

Since X, Y, Z are the same components of the heart vector as those occurring in equation (2) for WR, we may substitute the last three equations in those for WR. After a very elementary calculation we find: WR = (54a − 12b − 10c) LR + (8a + 40b − 6c) BR + (16a + 46b + 26c) FR, or in brief:

$$WR = p\ LR + q\ BR + r\ FR. \qquad (4)$$

In this formula, during the heart beat, WR, LR, BR, and FR change with time, while the quantities between brackets (p,q,r) remain constant. Thus it appears that the voltage of W with regard to R is a linear function of the three leads LR, BR, and FR used in the system B_1. This linear relation holds during the whole heart beat. It should be borne in mind that this theorem is of value only when the electrical activity of the heart can be described as that of a single dipole, wherever the latter may be situated.

Whether a linear relation as indicated by the last equation actually exists can be verified on a subject. For that purpose we used the earlier described universal vectorcardiograph (Becking, Burger, and van Milaan[1]).

The voltages LR, BR, and FR are amplified in an adjustable way and the results thus obtained are added up (with + or − sign). This total voltage is used to make the spot on the screen of a cathode ray tube deviate horizontally. The voltage WR (after amplification) is used to make this spot deflect vertically. If the quantities between brackets, (54a − 12b − 10c) = p, etc., in the linear combination (second member of equation[4]), have been chosen correctly, that is, in such a way that (4) has been fulfilled, then the horizontal and vertical deviations of the spot are equal at any moment, and the spot on the screen of the cathode ray tube moves over a straight line which makes an angle of 45° with the horizontal and vertical directions.

If the factors p, q, r have not been chosen correctly a curve is seen on the screen, which, just as a projection of a vectorcardiogram, consists of P, QRS, and T loops. It is now tried, by turning the knobs of the instrument (and possibly changing the sign of one or two of the factors) to reduce these loops to one line under 45°. We did this with ninety-six subjects with normal hearts. With some of them we succeeded very well in reducing the loops to the desired line. A case may occur in which with different combinations of the factors nearly as reasonable a result is obtained. This may be connected with the form of the vectorcardiogram. We will not go further into this. By these means determination of the value of the three factors becomes inaccurate in these cases.

It may also be found that one has not succeeded in making the loop resemble a straight line in a satisfactory way. Of course then the determination of the factors also is uncertain.

Once the three factors p, q, r of a subject have been determined, the observation on the subject will have illustrated that for him, during the heart beat, the equation

$$WR = p\ LR + q\ BR + r\ FR \qquad (5)$$

holds.

In this formula p, q, and r are numerically known. Comparison of (5) with (4), which have to hold for all time, that is, for all kinds of values of LR, BR, and FR, informs us that then the factors in (4) and (5) must be equal:

$$\begin{aligned} 54\ a - 12\ b - 10\ c &= p \\ 8\ a + 40\ b - 6\ c &= q \\ 16\ a + 26\ b + 26\ c &= r \end{aligned} \qquad (6)$$

In these equations p, q, r have been found experimentally and are therefore known. Three unknowns, a, b, c, can be calculated from them. As has been remarked already these are the required coordinates of the image point of the electrode position W. The result in the ninety-six subjects is presented in Figs. 1 and 2. In these figures in horizontal and frontal projection these points are shown and moreover the tetrahedron LRFB of the system B_1 is represented.

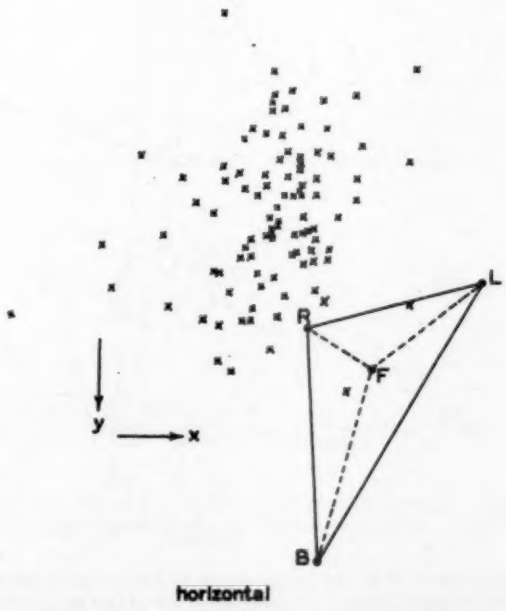

Fig. 1.—Place of the back electrode W in the image space in horizontal projection, determined according to the method of Becking. Each cross gives the result of the measurement on one subject. *R* = right arm, *L* = left arm, *F* = left leg, *B* = chest electrode. These form the tetrahedron in the system B_1.

It will be noticed that the scattering of the found points is of the order of the dimensions of the tetrahedron, and can certainly not be considered small. This is partly due to the errors in the determination of each of the points. But the scattering is certainly for the greater part real; there is a difference between different individuals.

The mean of all values found gives the coordinates a, b, c of the center of gravitation W in Figs. 1 and 2. These values of a, b, c are substituted in (2).

The system in which these coefficients are used we call W_4'. The components X, Y, Z of the heart vector expressed in LR, FR, and WR can be found from (3) and (2) by eliminating BR from these four relations.

$$W_4' \begin{cases} X = 53\ LR - 16\ WR + 15\ FR \\ Y = -24\ LR - 76\ WR + 23\ FR \\ Z = -\ 8\ LR + 13\ WR + 27\ FR \end{cases} \qquad (7)$$

VECTORCARDIOGRAMS WITH THE IMPROVED SYSTEM W_4'

With these coefficients we have recorded vectorcardiograms from many of the former subjects. For this purpose the previously described universal vectorcardiograph was used (Becking, Burger, and van Milaan[1]). The result was better than might have been expected on the ground of the scattering repre-

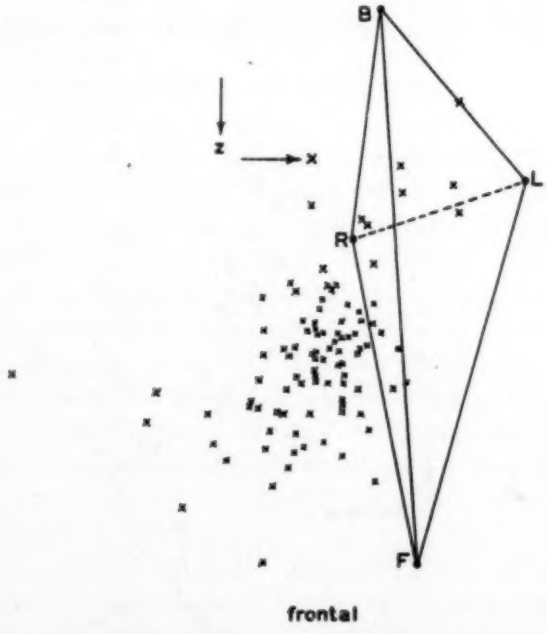

Fig. 2.—Place of the back electrode W in the image space in frontal projection, determined according to the method of Becking. Each cross indicates the result of the measurement on one subject.

sented in Figs. 1 and 2; the agreement of W_4' with B_1 was satisfactory. This holds not only for the frontal projection, for which the agreement is to be expected. Here the electrodes W and B, respectively, on back and chest play but a small part. These electrodes serve mainly to find the sagittal component that is decisive for the form of the horizontal projection. Also for the latter the agreement was better, however, than that between the original W_4 (according to Wilson and associates) and B_1.

FURTHER IMPROVEMENT OF THE SYSTEM OF COEFFICIENTS (W_4'')

Further consideration of the vectorcardiograms revealed that there is a slight systematic deviation between the horizontal projection of W_4' and B_1; the loops of W_4' must be rotated clockwise an average of 20° in order to get the direction of that of B_1. This rotation, together with a small enlargement of the loops in the X direction, leads to a correction of the coefficients of (7).

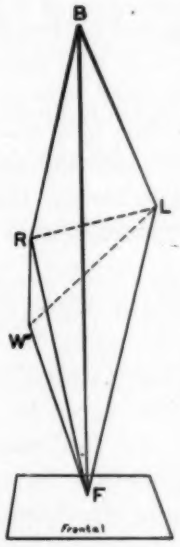

Fig. 3.—Tetrahedron BRLF and W''RLF of the systems B_1 and W_4''.

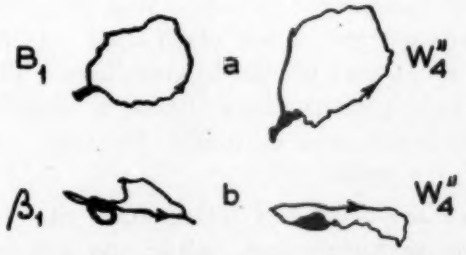

Fig. 4—*a*, Example of good agreement of the horizontal projection. Estimation mark 8. *b*, Example of bad agreement of the horizontal projection. Estimation mark 3.

The new coefficients are those of the following equations:

$$W_4'' \begin{cases} X = 58\ LR - 17\ WR + 16\ FR \\ Y = -\ 6\ LR - 81\ WR + 28\ FR \\ Z = -\ 8\ LR + 13\ WR + 27\ FR \end{cases} \qquad (7a)$$

The situation in the space image is represented by Fig. 3. In it we see the two tetrahedrons LRFB (system B_1) and LRFW (system W_4'') which have the frontal plane LRF in common. The first lies before this plane, the second behind it.

With the coefficients of (7a) vectorcardiograms were recorded (system W_4'') and compared with those according to system B_1 (cf. [3]). The agreement of the horizontal projections has indeed improved by the last correction. The frontal projection regularly yielded an excellent agreement, which was to be expected. Fig. 4 is an example of a good and a less good agreement.

Just as formerly, we (Burger, van Milaan, and den Boer[5]) have indicated the agreement of two projections with a mark, whereby 10 represents an ideal agreement and 0 the complete absence of it. The figures pertaining to the cases in Fig. 4 are stated in the legends.

CONCLUSION

The mean figure for sixty-four normal subjects is 6.4 ($\pm$ 0.2) and that for ninety-six patients 6.5 ($\pm$ 0.2), both for the horizontal projection. The vertical projection gives, as was to be expected, an even better agreement, namely 8.8 ($\pm$ 0.2).

The comparison of the original system of Wilson and associates with our system B_1 has earlier been given the mark 5.1 ($\pm$ 0.1) (Burger, van Milaan, and den Boer[5]). The application of the rationally chosen coefficients has thus led to a significant improvement in the agreement. In only about 10 per cent of the cases was the agreement of the horizontal projections in our estimation insufficient. For the clinical application the systems W_4'' and B_1 are easily in most cases to be considered as being equivalent.

DISCUSSION

The system of the equilateral triangle (W_4) proposed by Wilson and associates[6] has several advantages and is often used. It has, however, the disadvantage that it has no correct physical foundation. The system B_1 (Burger, van Milaan, and den Boer[5]), on the other hand, is based on physical principles and measurements, but is not used by many clinicians. A comparison of these two systems seemed worth while.

The measurements just described demonstrate that it is possible to obtain physically founded vectorcardiograms, while the electrode positions of the system of Wilson and associates are maintained. The coefficients of this new system (W_4'') are then to be chosen according to the equations (7a). The vectorcardiograms according to W_4'' give a far better agreement with our system B_1 than did the original system W_4 of the equilateral tetrahedron. In the majority of cases the agreement is clinically satisfactory.

The technical difficulties in applying physically founded coefficients are not serious. In a previous paper (Becking, Burger, and van Milaan[1]) a satisfactory method was described. Even a simpler way could be followed, though it will not be discussed here. It must be emphasized, however, that, once the instrument has been constructed, the clinician is no longer troubled with the application of "coefficients." In practice the use of a method with correct coefficients is just as simple as following a procedure of the "intuitive" type.

SUMMARY

The agreement of the system of the regular tetrahedron with the physically founded system B_1 is not very satisfactory in horizontal projection. This agreement becomes satisfactory and clinically useful by application of suitable coefficients, while maintaining the electrode places, as indicated by Wilson and associates.

SUMMARIO IN INTERLINGUA

Le accordo del systema del tetrahedro regular con le systema B_1 a fundamento physic (Burger, van Milaan, den Boer) es pauco satisfacente in le projection horizontal. Le accordo deveni satisfacente e clinicamente utile per le application de appropriate coefficientes durante que le sitos del electrodos es mantenite como Wilson e su associatos los indicava.

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ANALYSIS OF R,L,F,B SYSTEMS OF SPATIAL VECTORCARDIOGRAPHY

ERNEST FRANK, PH.D.

PHILADELPHIA, PA.

INTRODUCTION

THE human body is a three-dimensional conducting medium in which current fields produced by electrical activity of heart muscle change violently during the cardiac cycle causing a sequence of potential differences to appear in rapid undulation over the entire body surface. It would seem self-evident that a three-dimensional viewpoint must be taken in order to extract maximum information concerning electrical activity of the heart from body-surface potentials, especially because physical orientation and location of the heart differ widely among individuals. A variety of three-dimensional methods are available for studying surface potentials, extending from the most elaborate (complete exploration of the entire body surface by recording a great number of simultaneous potential differences) to the simplest (measurement of three judiciously chosen independent simultaneous potential differences). The most elaborate method obtains all data available from surface potentials but is generally impractical. The simplest method can be made very practical but gives limited information. Somewhere in this wide range of possibilities it is desirable to evolve a three-dimensional clinical method which strikes an optimum compromise among such factors as soundness of theoretical basis, accuracy, reproducibility, speed of application, cost of equipment, and others. The optimum method is not known today nor is there agreement on the relative emphasis to be placed on the varied factors mentioned. Since the vehicle of knowledge is sound theory, it seems plausible to concentrate on this aspect during the developmental period, especially because empirical methods have limited growth potential.

Spatial vectorcardiography, a method in the simplest category, represents one approach to this three-dimensional problem. Its foundation rests upon the assumption that heart activity may be represented by a fixed-location equivalent dipole, for with this assumption it can be shown[1] that complete information concerning equivalent dipole behavior is obtainable, in principle, from any three independent potential differences on the surface of the medium. A wide variety of systems of spatial vectorcardiography have been proposed. Because they

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do not yield accurate determinations of dipole components (as presently used) when applied to homogeneous three-dimensional torso models,[2] it is hardly to be expected that they would be satisfactory when applied to humans. However, the Wilson tetrahedron system, with suitable modification of standardizing factors, has been shown[2] to be capable of greater accuracy than some other commonly used systems for deducing from surface potentials the components of a fixed-location dipole in homogeneous three-dimensional torso models. Accuracy is critically dependent on dipole location,[3] and this constitutes a fundamental limitation not only for this system but for others presently in use.

Influence of dipole location on surface potentials has been discussed in detail for frontal-plane electrocardiography where effects are found to be very substantial.[3,4] Therefore, it is logical to expect sizeable variations to arise from this source in spatial vectorcardiography. Quantitative evidence supporting applicability of fixed-location dipole potentials on three-dimensional models to the QRS complex in the normal male subject[5-8] suggests that it is worthwhile and not unrealistic to investigate dipole-location effects in such models for vectorcardiography, and to present data which may be useful for approximate correction of sizeable errors. Consequently, basic physical data are given here for standard limb electrodes *R*, *L*, and *F* and a back electrode *B* for a range of dipole locations believed to encompass most individuals. These data are presented in a form that can be applied in practice, provided dipole location can be estimated accurately. A proved clinical basis for making this estimate with required accuracy is not presently available, although two different research methods for locating the dipole have been proposed.[6,9] Inherent vulnerability to dipole location of the modified Wilson tetrahedron and other systems of vectorcardiography which may be devised using electrodes *R*,*L*,*F*,*B* is illustrated by applying the basic data. It is concluded that accurate results are unattainable in vectorcardiography if equivalent dipole location is ignored.

EXPERIMENTAL METHOD

Potentials at right arm (*R*), left arm (*L*), left leg (*F*), and back (*B*) were measured with respect to the mid-potential of a finite dipole immersed in accurate, life-size, three-dimensional male and female homogeneous torso models using methods previously outlined in detail.[3,10] For each model, 27 different dipole locations (indicated in Fig. 1) were employed to cover a range believed to be reasonable for the human heart in various individuals. The rectangular volume shown in Fig. 1 was located with respect to right and left mid-axillary lines and front and back midlines so that data presented may be applied to a variety of individuals using anatomic landmarks which are convenient. The back electrode (*B*) was fixed permanently on each torso model directly behind dipole location *O*; to the left of the back midline by an amount equal to 10 per cent of the thorax width (3.3 cm. for the male torso; 2.5 cm. for the female torso). Data for back electrodes located precisely behind each of the 27 dipole locations were also obtained but are not presented for reasons given in the discussion. Dipole orientations along the standard[11] *x*,*y*, and *z* axes of Fig. 1 were used for

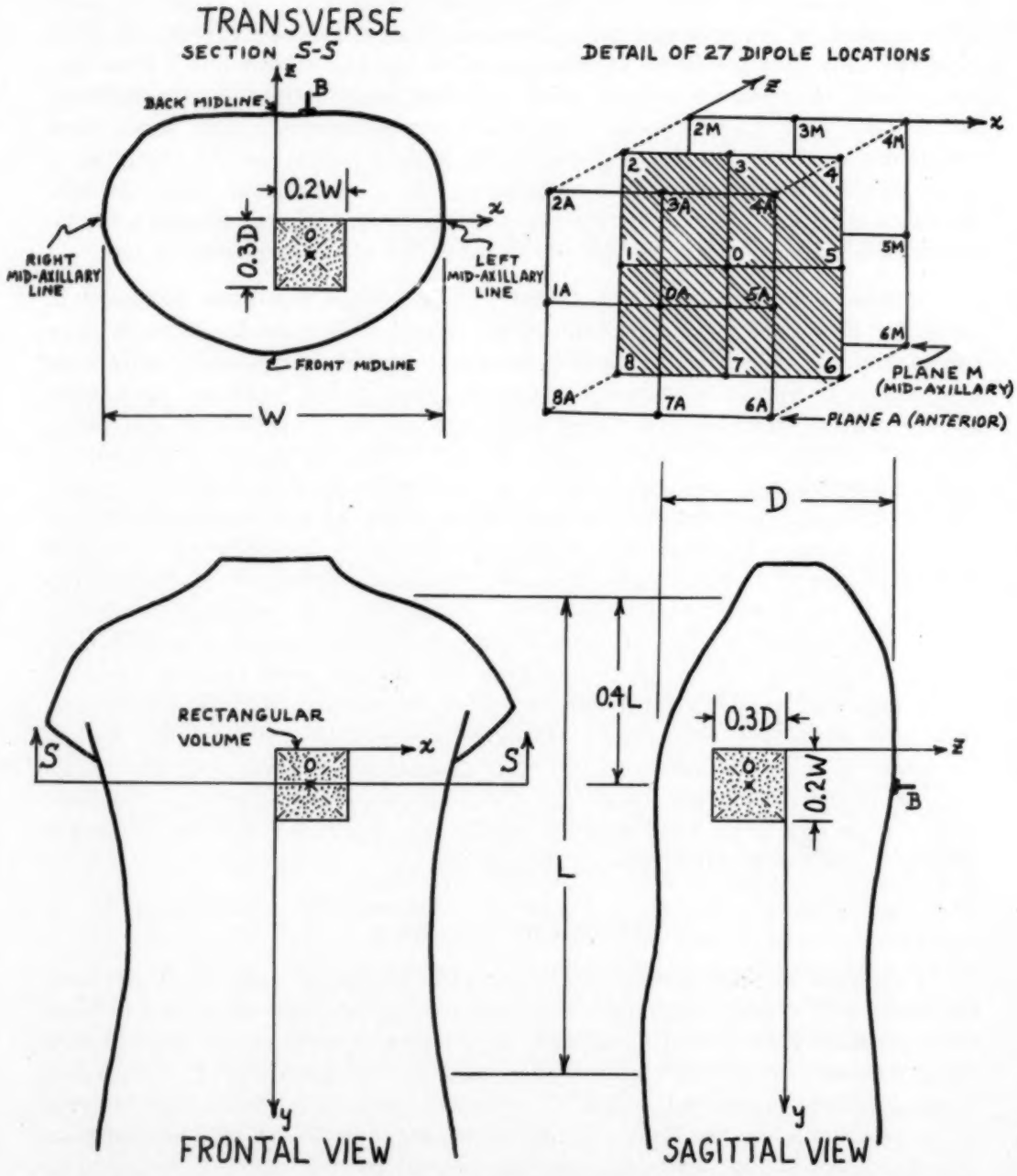


Fig. 1.—Three views of torso model showing rectangular volume containing dipole locations used experimentally for each torso. Rear face of rectangular volume coincides with plane containing right and left mid-axillary lines; right face of rectangular volume coincides with plane containing front and back midlines. The center of the volume (point *O*) is down approximately 40 per cent of the total distance from the xiphosternal notch to the iliac spine. A cross section of the thorax at the level of location *O* is shown in upper left, *W* = thorax width, *D* = thorax depth. Dimensions of the rectangular volume are expressed as fractions of *W* and *D* to permit flexible application of coefficients in Fig. 2 for a variety of individuals. (For male model, *W* = 33 cm., *D* = 25 cm.; for female model *W* = 25 cm., D = 18 cm.) Dipole locations at 27 points, enlarged in upper right, are identified by clockwise numbering in each of three planes parallel to the frontal plane. Number locations are followed by letter *A* in anterior plane and by *M* in plane of mid-axillary lines.

each model to obtain three unipolar coefficients[1] associated with each boundary electrode for each dipole location (648 coefficients) with an accuracy of approximately 5 per cent. Detailed procedure has been presented elsewhere.[3]

RESULTS

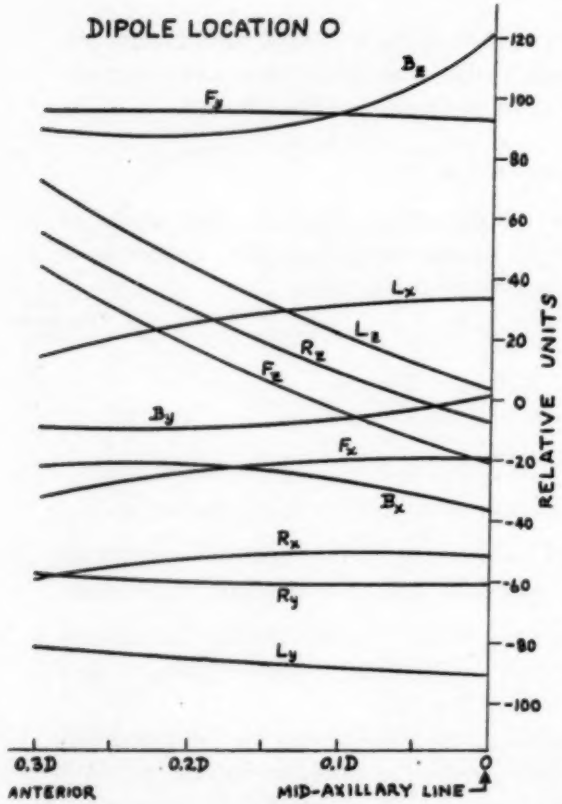
Results may be expressed in terms of equations relating true unipolar electrode potentials to dipole components using experimentally determined coefficients for each dipole location.[1] Equations are in the form

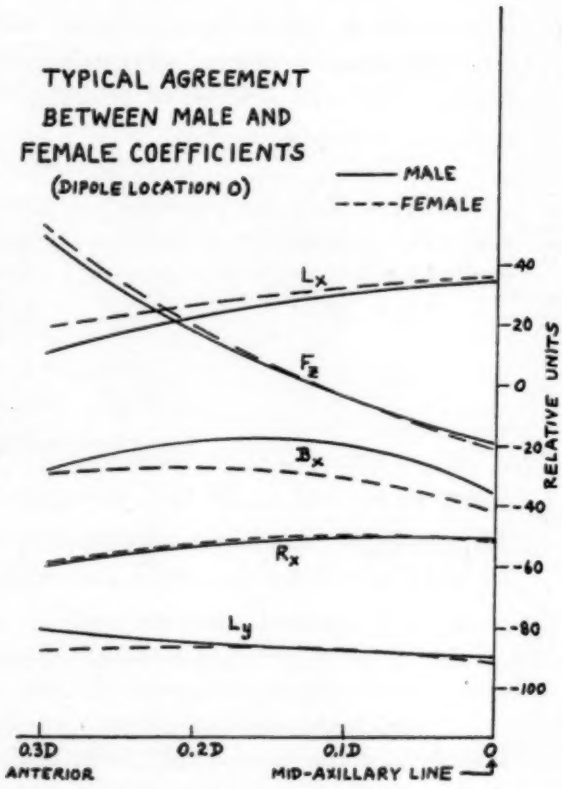
$$\begin{aligned} V_R &= R_x p_x + R_y p_y + R_z p_z = \vec{R} \cdot \vec{p} \\ V_L &= L_x p_x + L_y p_y + L_z p_z = \vec{L} \cdot \vec{p} \\ V_F &= F_x p_x + F_y p_y + F_z p_z = \vec{F} \cdot \vec{p} \\ V_B &= B_x p_x + B_y p_y + B_z p_z = \vec{B} \cdot \vec{p} \end{aligned} \qquad (1)$$

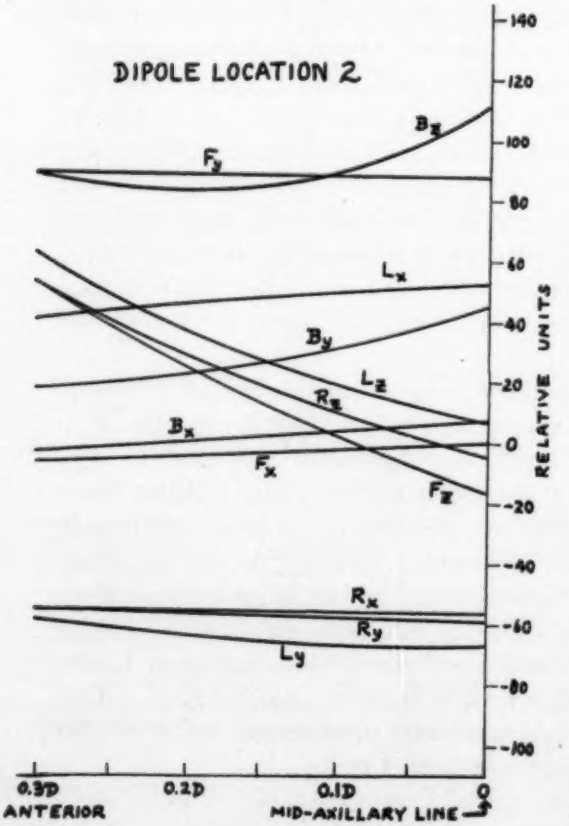
where p_x,p_y, and p_z are the rectangular components of the dipole vector $\vec{p}$; R_x, R_y, and R_z are experimentally determined coefficients (components of vector $\vec{R}$) for electrode R; L_x, L_y, and L_z are experimentally determined coefficients (components of vector $\vec{L}$) for electrode L; and so forth. True unipolar potentials at electrodes R,L,F,B may be calculated for any arbitrary dipole strength and orientation using equations of the form given in Eq. (1).

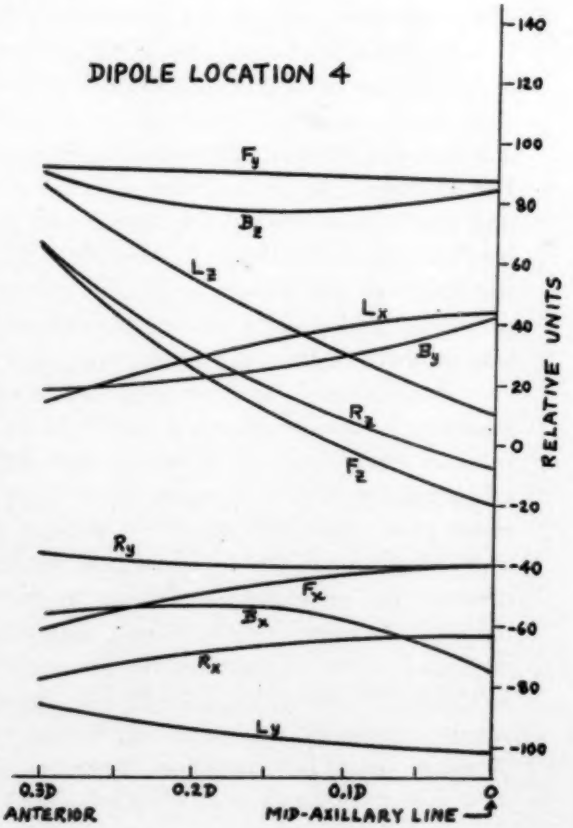
Coefficients determined for the female torso were found to be uniformly larger than the male-torso coefficients because of the smaller female physical dimensions.[3] Since only relative coefficients are of interest, all female coefficients were reduced by a common factor for comparison with male results. It was found that male and reduced female coefficients agreed within ± 10 units or better for corresponding dipole locations (example in Fig. 2) thus revealing that dipole location expressed as a fraction of thorax dimensions (as in Fig. 1) is a meaningful way of comparing eccentricity in systems of different dimensions. For example, dipole location *4M*, 5.0 cm. left of the plane containing the front and back midlines in the female torso, yielded essentially the same reduced coefficients as the male torso location *4M*, which was eccentric by 6.6 cm. Since the shape of the female torso differed considerably from the male, this reflects a property that has been emphasized previously[3,6]; namely, torso contour is far less important than dipole eccentricity.

Because male and reduced female coefficients agreed closely for all 27 dipole locations, average of male and female results, given graphically in Fig. 2 in relative units, may be taken as working coefficients for most body builds. Coefficients for dipole location *O*, a typical ventricle center, differ slightly from those previously published[1,3,12] because they are the average of male and female and also because the location *O* is slightly different. Coefficients are not given directly for all dipole locations, to conserve space, because intermediate values may be obtained with sufficient accuracy by linear interpolation from the curves in Fig. 2. For instance, if L_x for location 1A is desired, the average of L_x for locations 8A (20 units) and 2A (42 units), which is 31 units, may be used. This result compares favorably with the experimentally determined value of 34. Average single interpolation errors are approximately 4 units.


DIPOLE LOCATION O
B_z
F_y
L_x
L_z
R_z
F_z
B_y
F_x
B_x
R_x
R_y
L_y
120
100
80
60
40
20
0
-20
-40
-60
-80
-100
RELATIVE UNITS
0.3D
0.2D
0.1D
0
ANTERIOR
MID-AXILLARY LINE


TYPICAL AGREEMENT
BETWEEN MALE AND
FEMALE COEFFICIENTS
(DIPOLE LOCATION O)
MALE
FEMALE
L_x
F_z
B_x
R_x
L_y
40
20
0
-20
-40
-60
-80
-100
RELATIVE UNITS
0.3D
0.2D
0.1D
0
ANTERIOR
MID-AXILLARY LINE


DIPOLE LOCATION 2
B_z
F_y
L_x
B_y
L_z
R_z
B_x
F_x
F_z
R_x
R_y
L_y
140
120
100
80
60
40
20
0
-20
-40
-60
-80
-100
RELATIVE UNITS
0.3D
0.2D
0.1D
0
ANTERIOR
MID-AXILLARY LINE


DIPOLE LOCATION 4
F_y
B_z
L_z
L_x
B_y
R_z
F_z
R_y
F_x
B_x
R_x
L_y
140
120
100
80
60
40
20
0
-20
-40
-60
-80
-100
RELATIVE UNITS
0.3D
0.2D
0.1D
0
ANTERIOR
MID-AXILLARY LINE

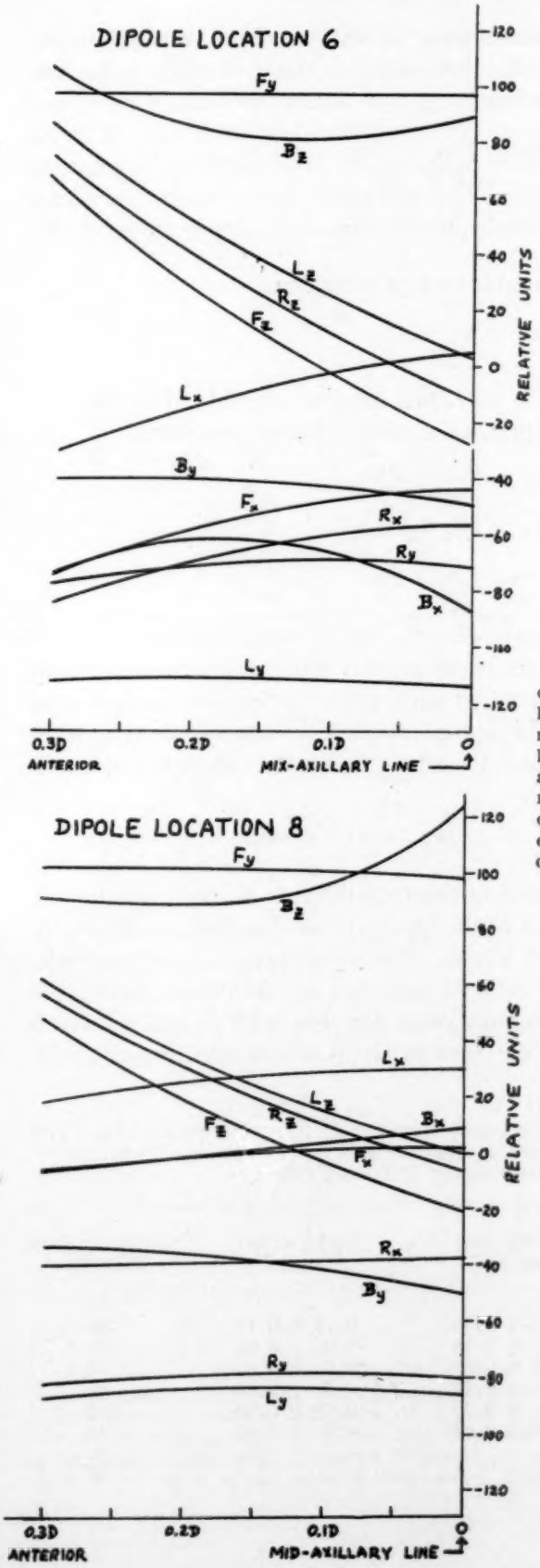


Fig. 2.—Average male and female torso model coefficients for dipole locations within rectangular volume of Fig. 1. Symbols on each curve refer to coefficients given in Eq. (1). For each location number, variation of coefficients with anterior dipole movement from the plane of the mid-axillary lines by an amount equal to 30 per cent of the thorax depth is given. Typical differences between male and reduced female coefficients are illustrated for dipole location *O*.

Coefficients for various bipolar leads may be derived from Eq. (1) using the unipolar coefficients given in Fig. 2. For example, Lead $I = V_L - V_R$ for dipole location O is obtained by subtracting

$$V_R = -50p_x - 60p_y + 20p_z$$

from

$$V_L = 29p_x - 86p_y + 34p_z$$

resulting in

$$V_L - V_R = 79p_x - 26p_y + 14p_z.$$

Wilson central-terminal leads are obtainable by first evaluating

$$V_C = \frac{1}{3}(V_R + V_L + V_F)$$

from Eq. (1) for a given dipole location and then subtracting this from the unipolar potential in question. For example, in the case of dipole location O,

$$V_C = -14p_x - 17p_y + 21p_z$$

and

$$V_B = -22p_x - 8p_y + 90p_z;$$

therefore,

$$V_B - V_C = -8p_x + 9p_y + 69p_z.$$

Approximate limits of variation of coefficients for all 27 dipole locations within the rectangular volume of Fig. 1 and for some bipolar leads which are employed in practice are given in Table I, normalized such that the largest coefficient of the three for each lead is unity. The actual coefficients are obtainable from Table I by multiplication of the fractional coefficients by the tabulated normalization factor. For example,

$$V_L = (80 \pm 25)p_x - (26 \pm 28)p_y + (12 \pm 8)p_z.$$

It may be seen that the relative variation of these coefficients is very pronounced, except in the cases of $V_F - V_C$ which is the least vulnerable lead to dipole location, and $V_F - V_R$ = Lead II which is almost as invulnerable. For example, $V_B - V_C$ displays limits of variation of ± 81 per cent in coefficients associated with p_z and has almost 20 per cent nominal contributions from p_x and p_y which can vary by ± 47 per cent and ± 61 per cent of the p_z coefficient, respectively.

TABLE I. LIMITS OF VARIATION OF COEFFICIENTS OF VARIOUS *R, L, F, B* BIPOLAR LEADS FOR DIPOLE LOCATIONS WITHIN RECTANGULAR VOLUME OF FIG. 1 EXPRESSED AS A FRACTION OF THE LARGEST COEFFICIENT OF EACH LEAD

LEAD	COEFFICIENT OF p_x	COEFFICIENT OF p_y	COEFFICIENT OF p_z	NORMALIZATION FACTOR
$V_L - V_R$	1.0 ± 0.31	−0.32 ± 0.35	0.15 ± 0.10	80
$V_F - V_R$	0.19 ± 0.14	1.0 ± 0.18	−0.05 ± 0.04	156
$V_B - V_R$	0.34 ± 0.67	0.80 ± 0.59	1.0 ± 0.64	70
$V_R - V_C$	0.86 ± 0.40	−1.0 ± 0.44	0 ± 0.10	43
$V_F - V_C$	0.06 ± 0.10	1.0 ± 0.13	−0.09 ± 0.04	113
$V_B - V_C$	−0.17 ± 0.47	0.18 ± 0.61	1.0 ± 0.81	70

While certain variations in coefficients may be seen graphically in Fig. 2 and numerically in Table I, it is perhaps helpful to examine these effects in image space, the basis for which has been discussed elsewhere.[1,12] Three cases are given in Fig. 3 for the most extreme deviations among the 27 locations. Coefficients are compared in image space with location *O* for locations *2M* and *6A* which are at diagonally opposite corners of the rectangular volume. General trends of the tetrahedrons in image space observed for all 27 dipole locations are

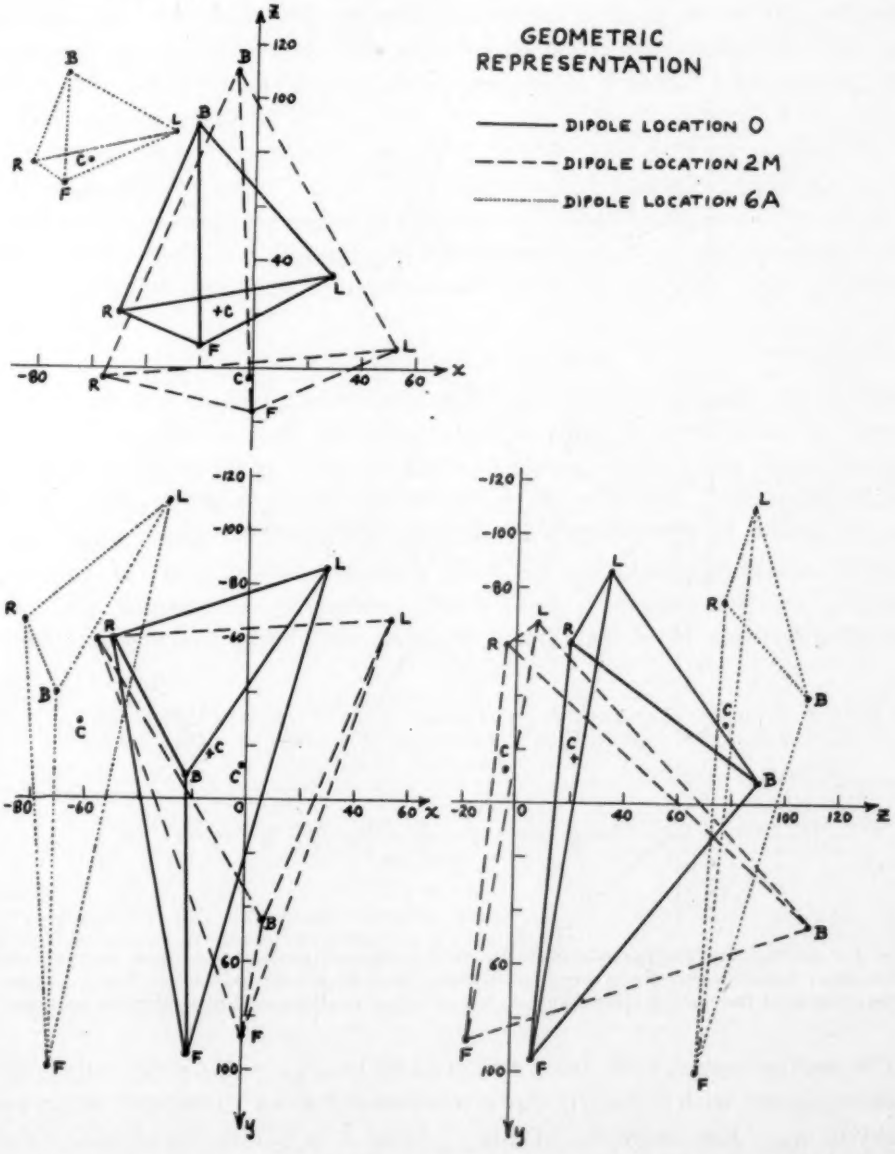


Fig. 3.—Geometric representation of electrodes *R*, *L*, *F*, *B* in image space for three dipole locations. Locations *2M* and *6A*, at diagonally opposite corners of the rectangular volume of Fig. 1, give greatest departures from conditions at location *O*. Wilson central-terminal is represented in image space by point *C*, the median point of the limb-lead triangle *R*, *L*, *F*. Coordinate system origin corresponds to the dipole mid-potential. Potential differences are equal to the dot (scalar) product of heart dipole vector $\vec{p}$ and vector joining points in image space. Discussion in text.

typified in the three cases selected in Fig. 3. The vector from *B* to *C*, from which $V_B - V_C$ is derivable, is reduced sharply in length when the dipole location is moved anteriorly because the frontal plane shifts toward the rear in image space. For the range of locations studied, this effect results in a drastic amplitude variation of more than 5:1 which means that, aside from shape errors, the required standardization factor for $V_B - V_C$ can vary by this amount. Shape errors of $V_B - V_C$ may also be seen to be substantial from Fig. 3 since the direction of the vector from *C* to *B* shifts upward as the dipole is moved forward and leftward.

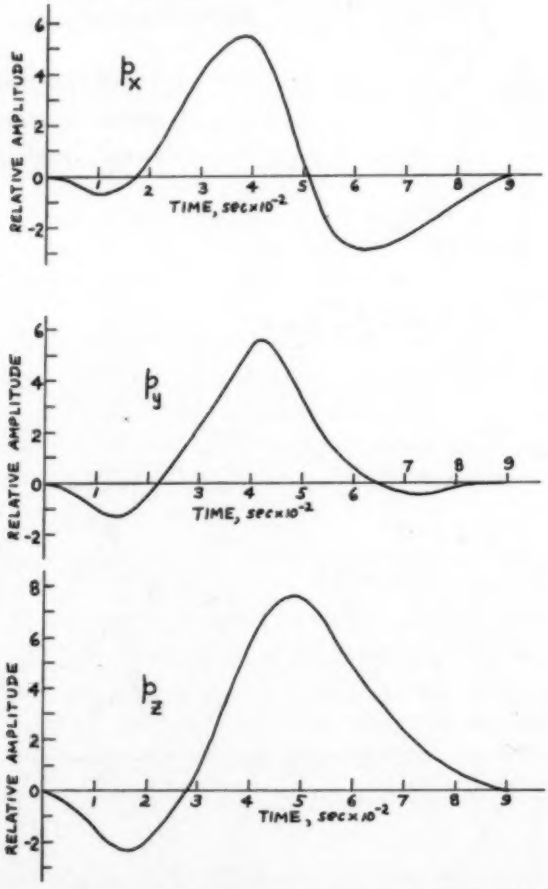


Fig. 4.—Rectangular components of dipole vector assigned to illustrate dipole location effects for a specific case. Dipole vector loops are given in three views by the dotted lines in Fig. 7. These dipole functions, typical of the normal QRS complex, are the same as those used in a previous analysis.[2]

The vector from *L* to *R*, from which Lead I = $V_L - V_R$ is derivable, changes direction greatly with leftward dipole movement so as to contain an increasing amount of p_y. For example, in Fig. 3 Lead I is nearly horizontal for dipole location *2M*, since this location is symmetric in the torso, but slants upward by about 40° for eccentric location *6A*. This means that the shape of Lead I is strongly dependent on dipole location and cannot be generally relied upon to yield a shape faithful to p_x. Moreover, the length of the vector from *L* to *R* generally increases as the dipole is moved upward. This is also evident in Fig.

3 and indicates that the amplitude of Lead I increases for dipole locations higher in the chest. An amplitude range of 3:1 is found for dipole locations employed in this study.

The direction and magnitude of the vector from *F* to *C*, from which $V_F - V_C$ is derivable, tend to be fairly uniform for all dipole locations studied. This is the best lead from the standpoint of invulnerability to dipole location changes. Even so, its amplitude ranges over about 1.5:1. Other trends may also be observed, but the major ones have been mentioned. Since potential differences are the measured quantities, shifts in location of the tetrahedron with respect to the dipole at the origin of image space are of no consequence; it is only the relative locations of the image points *R,L,F,B* with respect to each other that are significant.

No matter what viewpoint is taken (numerical plots of coefficients as in Fig. 2, tabular data as in Table I, or image space representation as in Fig. 3) the same quantitative conclusion is reached: Dipole location exerts a first-order effect on coefficients, and this impairs substantially the accuracy in shape, timing, and amplitude of dipole components deduced from systems that ignore dipole location effects.

ILLUSTRATIVE EXAMPLES

Although the core of the matter resides in the behavior of the coefficients, dipole location effects may be illustrated in more direct terms by assigning typical variations to p_x, p_y, and p_z and calculating from them bipolar leads and vectorcardiograms that would be observed for different dipole locations but the same dipole behavior. Dipole components shown in Fig. 4, which are reasonably similar to those associated with the QRS complex in normals, have been adopted for illustrative purposes. Once these are assigned, specific instantaneous QRS surface potentials may be calculated from the coefficients presented.

TABLE II. EXAMPLE OF VARIATIONS IN AMPLITUDE, SHAPE, AND TIMING OF SCALAR QRS COMPLEX LEADS OF WILSON TETRAHEDRON FOR DIPOLE LOCATIONS WITHIN RECTANGULAR VOLUME OF FIG. 1

QRS COMPLEX CHARACTERISTIC	$V_F - V_C$	$V_L - V_R$	$V_B - V_C$
Amplitude:			
Per cent peak-to-peak amplitude variation about mean	±22	±50	±68
Ratio of maximum to minimum peak-to-peak amplitude	1.6	3.0	5.3
Per cent peak amplitude variation of Q wave about mean	±21	±78	±47
Per cent peak amplitude variation of R wave about mean	±22	±55	±71
Per cent peak amplitude variation of S wave about mean	±68	±40	
Timing:			
Milliseconds variation for peak of Q	± 1	± 1	± 5
Milliseconds variation for peak of R	± 1	± 3	± 5
Milliseconds variation for peak of S	± 1	± 3	
Limits of base line crossover between Q and R, milliseconds	± 1	± 3	± 6
Limits of base line crossover between R and S, milliseconds	± 3	± 3	

The three bipolar leads used in the Wilson "equilateral" tetrahedron were calculated for the dipole functions of Fig. 4, using dipole locations at the eight corners of the rectangular volume and for the center location *O*. Results are

given in Fig. 5 where variations in amplitude and shape for this particular dipole behavior resulting purely from dipole location changes may be seen qualitatively. Dipole behavior itself was identical at all locations. Results of detailed quantitative analysis of the nine QRS complex scalar leads in each case are summarized in Table II. Study of this table reveals very significant variations in amplitude, shape, and timing as a function of dipole location. It is obvious that if these surface potentials are all processed in the same fashion, ignoring dipole location, heart-vector components substantially different from the actual p_x, p_y, and p_z will usually be deduced.

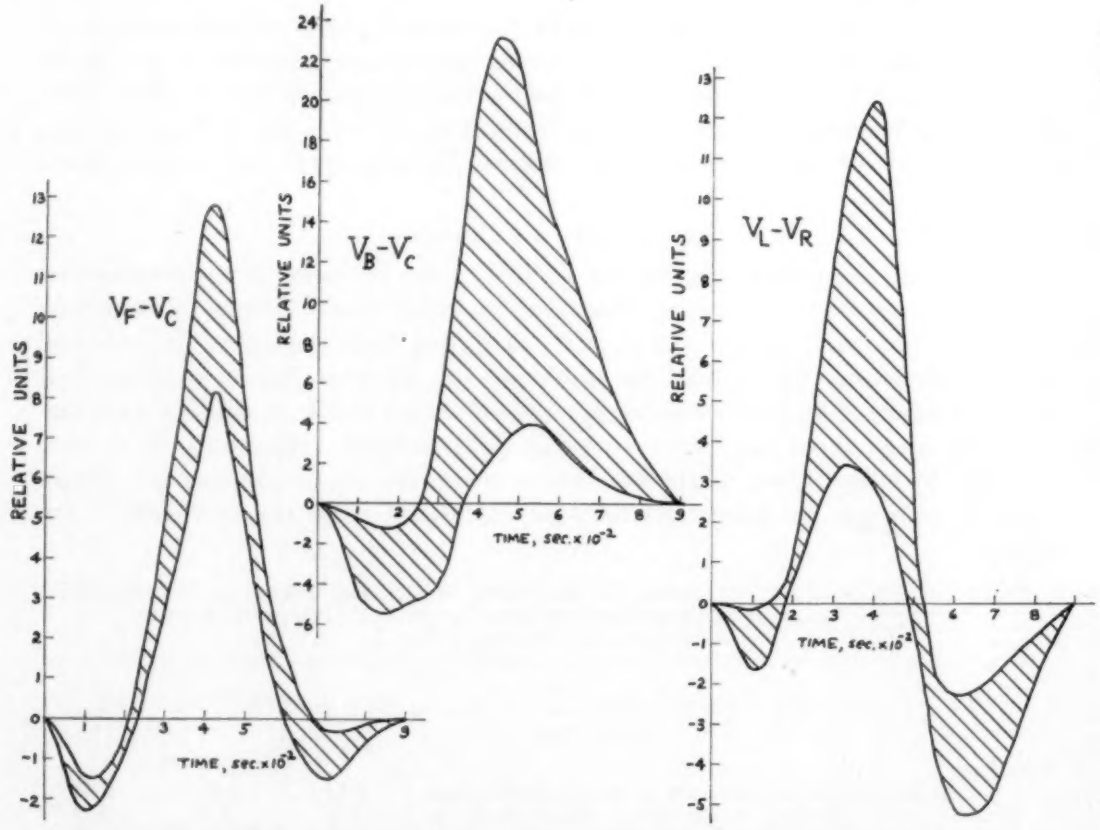


Fig. 5.—When dipole with components of Fig. 4 is varied in location within rectangular volume of Fig. 1, scalar leads of Wilson tetrahedron fall within shaded areas shown. Least susceptible lead is $V_F - V_C$. Quantitative details of scalar lead variations are given in Table II.

Vectorcardiograms derived from the Wilson tetrahedron with modified scale factors[2] are illustrated in Fig. 6 for the dipole behavior given in Fig. 4. These vectorcardiograms are obtained, for a given dipole location, by plotting $(V_L - V_R)$ vs. $\frac{3}{4}(V_F - V_C)$ for the frontal loop, $\frac{3}{4}(V_F - V_C)$ vs. $(V_B - V_C)$ for

the sagittal loop, and ($V_L - V_R$) vs. ($V_B - V_C$) for the transverse loop. When the dipole location is at *O*, the modified system yields quite a faithful result,[2] for this dipole behavior, as may be seen by comparison with the dipole loops

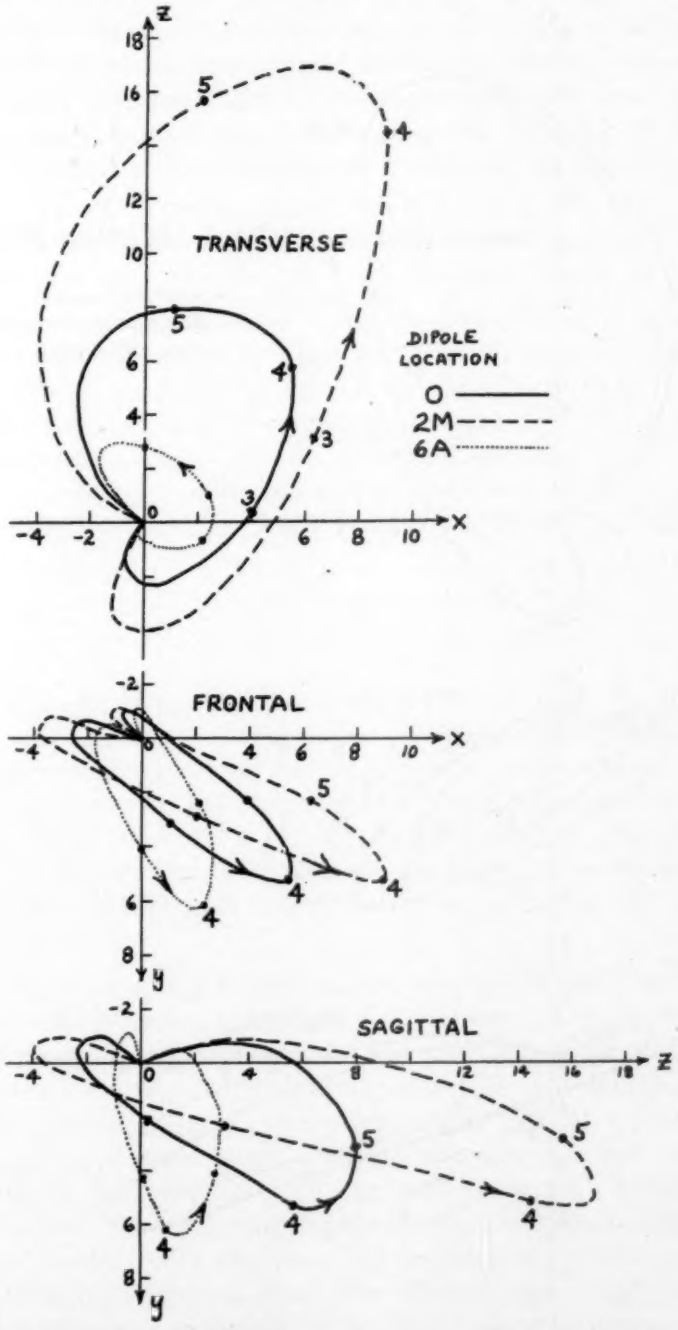


Fig. 6.—Vectorcardiograms of Wilson tetrahedron with modified scale factors are shown for three different dipole locations using the same dipole behavior of Fig. 4 at each location. For location *O*, the vectorcardiogram is in good agreement with dipole behavior (see Fig. 7), but substantial departures occur when dipole is moved to extreme locations *2M* and *6A* (See Fig. 3). Time in hundredths of a second is indicated by three points on each loop.

given in Fig. 7. However, pronounced deviations result when the dipole is moved to extreme corners of the rectangular volume, locations *2M* and *6A*, despite the fact that the dipole components themselves are unaltered. The

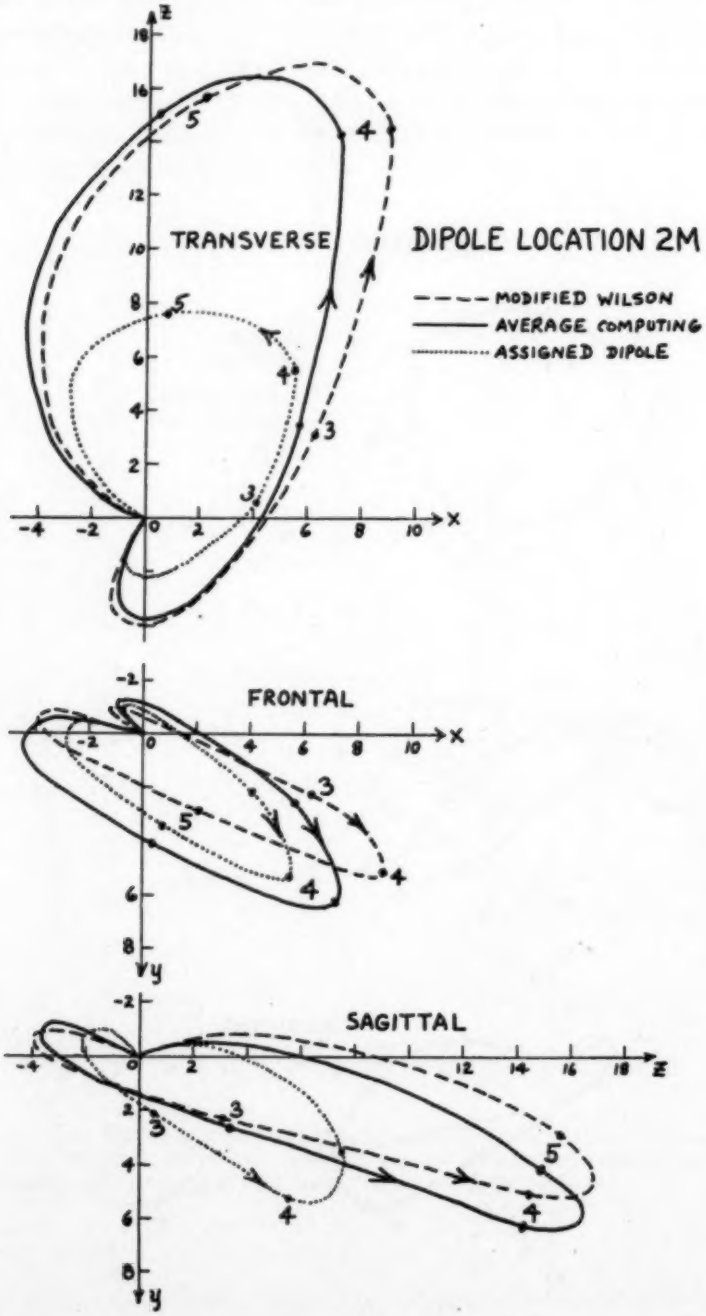


Fig. 7.—Comparison of vectorcardiograms derived from the Wilson tetrahedron and average computing system with assigned dipole loops, for dipole location *2M*. The average computing system (which agrees perfectly with dipole behavior at location *O*) may be seen to be somewhat less sensitive to dipole location changes, in this example, but still displays substantial errors. Time in hundredths of a second is indicated by three points on each loop.

two cases given represent the most adverse results for the rectangular volume studied; other locations produce less drastic departures from the results at location *O*. It should be noted that $\frac{3}{4}$ $(V_F - V_C)$ is considerably less sensitive to dipole location changes than the other two potential differences, as pointed out earlier. These vectorcardiograms combine the timing and amplitude variations which have been presented for the individual scalar leads in Table II and Fig. 5 and provide another way of assessing the influence of dipole location. The Wilson "equilateral" tetrahedron using originally proposed[13] standardization factors of 1.0, $\sqrt{3}$, $\sqrt{3/2}$ (rather than 1.0, 3/4, 1.0 as in Fig. 6) is subject to much larger errors[2] for dipole location *O* for the case illustrated, upon which are superimposed additional variations similar to those present in Fig. 6.

A system of vectorcardiography using electrodes *R,L,F,B* has been employed by Burger and van Milaan[14] which has provisions in the electrocardiograph[15] for solving equations for p_x, p_y, and p_z from potential differences $V_L - V_R$, $V_F - V_R$, and $V_B - V_R$, using average torso-model coefficients. This will be called an "average computing system" in this paper. Equations to be solved may be obtained by introducing coefficients into Eq. (1) from Fig. 2, and then solving[1] for the dipole components. The result is, for dipole location *O*,

$$\begin{aligned} p_x &= 122\ (V_L - V_R) + 27\ (V_F - V_R) - 20\ (V_B - V_R) \\ p_y &= -26\ (V_L - V_R) + 55\ (V_F - V_R) + 15\ (V_B - V_R) \\ p_z &= -30\ (V_L - V_R) - 52\ (V_F - V_R) + 139\ (V_B - V_R) \end{aligned} \qquad (2)$$

where relative coefficients are given, using a value of unity for the determinant rather than its actual value of 931×10^3. These coefficients compare favorably with those determined by Burger and van Milaan[16] in heterogeneous torso-model experiments, whose results are [followed by corresponding values from Eq. (2)]: 102(122), 28(27), −30(−20), −14(−26), 51(55), 24(15), 11(−30), −52(−52), 150(139). The largest disagreement is in the $V_L - V_R$ coefficient of p_z, which is suppressed to a considerable extent by the much larger size of the $V_B - V_R$ coefficient of p_z.

The average computing system differs from the Wilson tetrahedron in two ways: No central terminal is employed, and more refined equations are used to derive the heart-dipole components. For example, in the Wilson system the equation $p_x = k(V_L - V_R)$, where k is a constant, is used[2] rather than one which contains two correction terms as in the first line of Eq. (2). The fidelity of this average computing system, which employs the same average coefficients irrespective of dipole location, has been analyzed in detail. Variations in scalar leads $V_F - V_R$ and $V_B - V_R$ with dipole location are comparable to those obtained in Fig. 5 for $V_F - V_C$ and $V_B - V_C$, respectively, as may be seen in Table I. Vectorcardiograms derived from Eq. (2) are somewhat less sensitive to dipole-location changes, as illustrated in Fig. 7 for an extreme case, owing to more refined processing of the scalar leads in the equipment. Indeed, agreement with p_x, p_y, and p_z for dipole location *O* is perfect, in principle, since coefficients in Eq. (2) pertain to that location. However, large dipole location errors are

still present in this system and justification for use of the more elaborate equipment may be questioned when average coefficients, the same for all individuals, are used. The difficulty does not reside in the use of an improper average set of coefficients; indeed, coefficients for location *O* differ in no significant way from the average of those for all 27 locations. The basic source of error is that the coefficients themselves change so markedly with different dipole locations.

An "exact" computing system utilizing equipment similar to Burger's can be proposed,[17] since provisions for variable coefficients in the electrocardiograph are easily made by rotatable knobs. Dipole-location effects could be circumvented in such a system by introducing coefficients suitable for the individual subject being measured, and in principle the results would be as reliable as the coefficients. However, individualized coefficients for a given subject are not readily obtainable but quick, approximate methods might be established in the future. In an "exact" computing system there is no need or advantage whatever in using a central terminal or, indeed, in using any particular sets of bipolar leads among *R,L,F,B* as long as three different leads are used. The basic reason for this is that the four electrodes *R,L,F,B* provide three and only three independent potential differences. These are just sufficient to determine p_x, p_y, and p_z and there is no redundancy. The potential difference between any junction of any arrangement of resistors connected to *R,L,F,* or *B* provides no additional independent information. (This situation is analogous to the case in frontal-plane electrocardiography where electrodes *R,L,F* provide two independent potential differences, and no additional basic information is obtained from "unipolar" limb leads.) To illustrate, an "exact" computing system can be proposed using three "unipolar" leads from *R,F,* and *B* to the Wilson central terminal. Equations for such a system, for dipole location *O*, are

$$\begin{aligned} p_x &= -257(V_R - V_C) - 97(V_F - V_C) - 22(V_B - V_C) \\ p_y &= -\ 17(V_R - V_C) + 82(V_F - V_C) + 15(V_B - V_C) \\ p_z &= -\ 27(V_R - V_C) - 22(V_F - V_C) + 140(V_B - V_C). \end{aligned} \qquad (3)$$

Results computed from this system are identical to those obtained from Eq. (2) which does not employ a central terminal. In fact, Eqs. (2) and (3) may be derived one from the other. While there can be an accuracy improvement resulting from the use of a central terminal in a system such as the Wilson tetrahedron, as discussed elsewhere,[2] it is pointless to incorporate such a terminal in computing type systems.

DISCUSSION

It has been shown for homogeneous three-dimensional torso models with an immersed dipole that dipole location exerts a pronounced influence on *R,L, F,B* systems of spatial vectorcardiography. The applicability of these results to the human subject is surprisingly good for the QRS complex of one normal subject, where human and torso-model results agreed within ± 15 per cent.[7,8] It seems plausible from this to expect comparable applicability for most normals, since the experiments did not rely on any particular characteristics of the individual tested, but applicability to abnormal cases or to P or T waves has not yet

been established. Whether the model is homogeneous or heterogeneous appears to be of little consequence since coefficients in both cases are in reasonably close agreement. However, conduction disturbances in the human heart could impair the quantitative applicability of the dipole concept, undermining the basic assumption of vectorcardiography and the applicability of the analysis presented. Assuming the dipole representation is applicable, the least reliable aspect of the data which have been presented is probably the left-arm coefficients. These coefficients may be expected to vary considerably from one individual to another because of the steep potential gradient[7,8] at the left shoulder, owing to the leftward and forward location of most hearts. It is felt that effects of different left-shoulder structures among various individuals are not accurately correctable in any system of electrocardiography which employs the left arm.

Although the back electrode *B* was fixed on each model for all of the results presented, other back electrode locations were studied in search of a more accurate anteroposterior lead. Specifically, a "tailored" back electrode placed directly behind the dipole anatomically for each different dipole location was investigated. Location of the back electrode is somewhat critical, in general, and it was found that this particular method of electrode placement did not successfully eliminate the undesired contribution of p_x from $V_B - V_C$. It was found that to accomplish this objective, and hence obtain a purer lead, the back electrode should be placed somewhat leftward of the dipole. It would be difficult to judge exactly where to place it on the human subject, so it was felt to be impractical, particularly in view of an additional objectionable feature which was observed. The amplitude of the "tailored" back electrode was found to be even more susceptible to dipole location changes in the anteroposterior direction than the fixed back electrode shown in Fig. 1. For example, while the fixed back electrode displayed about 5:1 amplitude variations in $V_B - V_C$ with anteroposterior dipole movement of 0.3D, the "tailored" back electrode yielded 9:1 amplitude variations.

There are many possible approaches to minimizing dipole-location errors. One method is to seek electrode sites which, because of their position, yield potential differences inherently insensitive to dipole-location effects. Of course other factors must be borne in mind in choosing electrode sites, such as reproducibility of electrode placement and selection of sites which yield substantial contribution from the dipole components to be determined. In following this approach, image surfaces[12] of homogeneous torso models for a wide variety of dipole locations were studied. It was found that the precordium is the most susceptible area to dipole-location effects, and therefore less desirable than the back for the case of obtaining a lead with a sizeable portion of p_z. Precordial electrode placement is also extremely critical. The head-foot component p_y is most conveniently obtained by the use of *R* and *F* electrodes which are among the least sensitive to dipole-location effects. In considering electrode reproducibility and backlog of empirical data, the left arm becomes a practical choice from which to obtain data containing a high percentage of p_x, but the left arm has disadvantages previously mentioned and also displays considerable sensitivity to dipole location. However, *R,L,F,B* are representative of electrode

locations which result in less vulnerability to dipole-location effects than many other anatomic points. Hence, the essential conclusions of this study cannot be altered in any great manner, quantitatively, by choice of better sites from the standpoint of dipole-location errors because *R,L,F,B* already represent nearly the optimum choice.

Another approach is to attempt to place electrodes on the subject in an individualized manner rather than using standard prescribed anatomic locations for all individuals. The "tailored" back electrode is an example of this approach. This method has doubtful promise for ultimate clinical use for several reasons: The correspondence between anatomic points and electrical image points is not simple and is strongly dependent on dipole location, the very item one would attempt to circumvent. Accurate estimates for placement of individualized electrodes cannot be made without a knowledge of the equivalent dipole location. Moreover, reproducibility of electrode placement for serial studies would pose difficulties. Errors in judging precisely where to locate individualized electrodes could amount to as much or more than those present with the use of standard electrode placement.

Since the influence of dipole location is so pronounced, it would be very useful to know just how much location variation exists among humans. One may expect, based on anatomic heart location, that is is comparable to the rectangular volume studied here, but there is scant information presently available on the location of the equivalent dipole and its correlation with anatomic location of the heart. This is, obviously, a pertinent area of research in view of the results presented here.* If location variations are not too great (smaller than considered here) it is conceivable that groups of multiple electrodes, rather than single electrodes, with correction networks designed to give a first-derivative compensation such as proposed by Schmitt[18] might be practical. Multiple electrodes of this type reduce the vulnerability to dipole-location effects, but their effectiveness appears to be limited to relatively small location changes. A new system of vectorcardiography of this type, currently under study in this laboratory, is theoretically capable of accommodating dipole locations within a $5 \times 5 \times 5$ cm. volume.

Finally, an obvious method for minimizing dipole-location errors is to use an "exact" computing system, as described, in which different coefficients for each individual are introduced. The method is practical and simple, since complications can be designed into the equipment, but the chief and by no means minor difficulty is the matter of determining the appropriate coefficients. By using the results presented here, it may be roughly stated that for each centimeter of error in estimate of dipole location there would be as much as 15 per cent error in dipole component determination. Thus, for satisfactory accuracy the dipole location would have to be known to within about 1 cm. The best-known method for accomplishing this[6] is impractically time-consuming for clinical use.

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*Heart-center determinations[6] on forty subjects have been carried out by Dr. George E. Seiden at the University of Pennsylvania for the QRS complex in both normals and patients. Thirty-five of the cases fall within a $4 \times 4 \times 4$ cm. volume centered on point *O* of Fig. 1.

It is not only *R,L,F,B* systems which are vulnerable to dipole-location errors, but others as well. Since dipole location effects are so pronounced, it is to be expected that overlap in results would occur for a variety of different systems in which dipole-location errors are manifested differently in each system. Hence, claims that similar results are obtained for a variety of systems proposed are not impossible if the definition of "similar" is relaxed sufficiently. For example, the vectorcardiograms in Figs. 6 and 7 may be said to be qualitatively similar but they represent drastic quantitative differences. Moreover, comparative studies of various systems using computing equipment in which coefficients are varied until best agreement is established[16] are of limited value, since two systems might show fair correspondence, as in Fig. 7, yet both of them might contain considerable absolute errors.

Justification for more accuracy than afforded by present methods of vectorcardiography may be questioned legitimately on a practical basis and, conceivably, improved accuracy may not be worthwhile. For example, if accurate methods were applied to numerous normals in which dipole location as well as other known sources of systematic error were taken into account, would the range of variability of dipole components be reduced over findings of present methods? It is known that much variability is traceable to ignored factors, but how much still remains inherent in individual human variability from one subject to the next is not known. . If individual variability is the dominant factor, there would be less reason for pursuing accurate methods. On the other hand, if accurate methods can be shown to reveal invariants of the equivalent heart dipole not previously recognized because of ignored factors known to influence the results, then they would be extremely useful, particularly in borderline cases, since cases of gross heart disorders are recognizable with systems known to be inaccurate. One approach to obtaining the answer to this vital question is to establish and apply accurate methods, and to then compare results with those obtained with present-day qualitative methods.

SUMMARY

1. Various systems of spatial vectorcardiography employing right arm (*R*), left arm (*L*), left leg (*F*), and back electrode (*B*) are studied for homogeneous, three-dimensional male and female torso models using an accurate unipolar measurement technique.

2. Influence of dipole location on accuracy of determination of dipole components is emphasized.

3. Quantitative data are presented for dipole locations representative of the range expected in human subjects.

4. Quantitative analysis of various systems is presented in terms of equations, geometric representations, scalar leads, and vectorcardiograms.

5. It is concluded that accurate results are unattainable in vectorcardiography, if dipole location is ignored.

6. Various approaches for minimizing dipole location errors are discussed.

7. While evidence exists in support of the quantitative applicability of these results for the normal QRS complex, its general applicability remains to be established.

SUMMARIO IN INTERLINGUA

Es investigate in terminos absolute le accuratessa de varie systemas de vectocardiographia spatial. Le systemas investigate emplea electrodos del bracio dextere, del bracio sinistre, del gamba sinistre, e del dorso. Le technica usate labora con precise mesurationes unipolar e es basate super mascule e feminin modellos del torso. Attention special es prestate a errores del location de dipolos. Le conclusion obtenite stipula—in tanto que resultatos derivate ab modellos del torso es valide—que equivalente componentes dipolar del corde human non es accuratemente determinabile si le location del dipolos es negligite. Es discutite mesuras pro reducer le errores del location dipolar.

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CORRELATION OF THE SPATIAL VECTORCARDIOGRAM AND THE ELECTROCARDIOGRAM IN RIGHT VENTRICULAR HYPERTROPHY

E. A. STERN, M.D., AND S. M. TENNEY, M.D.

ROCHESTER, N. Y.

THE electrocardiogram is often inadequate in the diagnosis of right ventricular hypertrophy (RVH). The classical diagnostic pattern of large R aV_R and large R waves in the right precordial leads usually does not occur until RVH is of marked degree. Lesser degrees may show borderline ECG patterns or may be entirely within normal limits. Conversely, there may occur ECG patterns which have certain similarities to the pattern of RVH, but which are not associated with this anatomical abnormality. These include infantile ECG patterns and pure rotations. Inasmuch as the routine ECG represents scalar activity of the frontal plane and a portion of the horizontal plane of the body, it is not surprising that, with gross changes of electrical heart position, there will be instances in which its information is limited. The spatial vectorcardiogram (SVCG) which more nearly approaches the total pattern of electrical excitation of the heart may be of greater value in such circumstances.

In an attempt to define the areas of usefulness of the SVCG compared to the ECG, thirty records in nineteen patients with chronic pulmonary disease, with and without cor pulmonale, and eight in patients with RVH of primary cardiac origin have been made. The correlation of clinical and/or pathologic finding, together with the ECG and SVCG constituted the present study. Representative cases from each major group are presented, and the generalities of interpretation and genesis of the pertinent ECG deflections in each discussed.

METHODS

The SVCG has been displayed on a short persistent cathode ray oscilloscope screen (DuMont 304A) and photographed with a Graflex camera, using superpanchro press-type B film. The bipolar leads from the patient were amplified with standard DC amplifiers (Sanborn) and then connected to the paired plates of the oscilloscope. The trace was interrupted with an audiofrequency oscillator at the rate of four hundred per second, inserted at the intensity modu-

From The Department of Medicine, University of Rochester, School of Medicine and Dentistry, Rochester, N. Y.

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lator. The trace was so deformed that the pointed ends of the dots were in the direction of the wave of excitation. The various projections of the cardiac vector were photographed in sequence while the patient was in a semiprone position. A routine standard ECG was obtained in each patient and was compared with the SVCG. Interpretation was correlated with the clinical and pathologic findings of the patient. The lead placements used in this study were those of Grishman and Scherlis.[1]

RESULTS

Table I summarizes briefly the correlation of the anatomical and clinical (including roentgenologic) data with the electrocardiographic and vectorcardiographic diagnosis of right ventricular hypertrophy in each case. It is clear that the SVCG is superior to the ECG on the whole in this regard. Pertinent representative cases to illustrate the significant features follow.

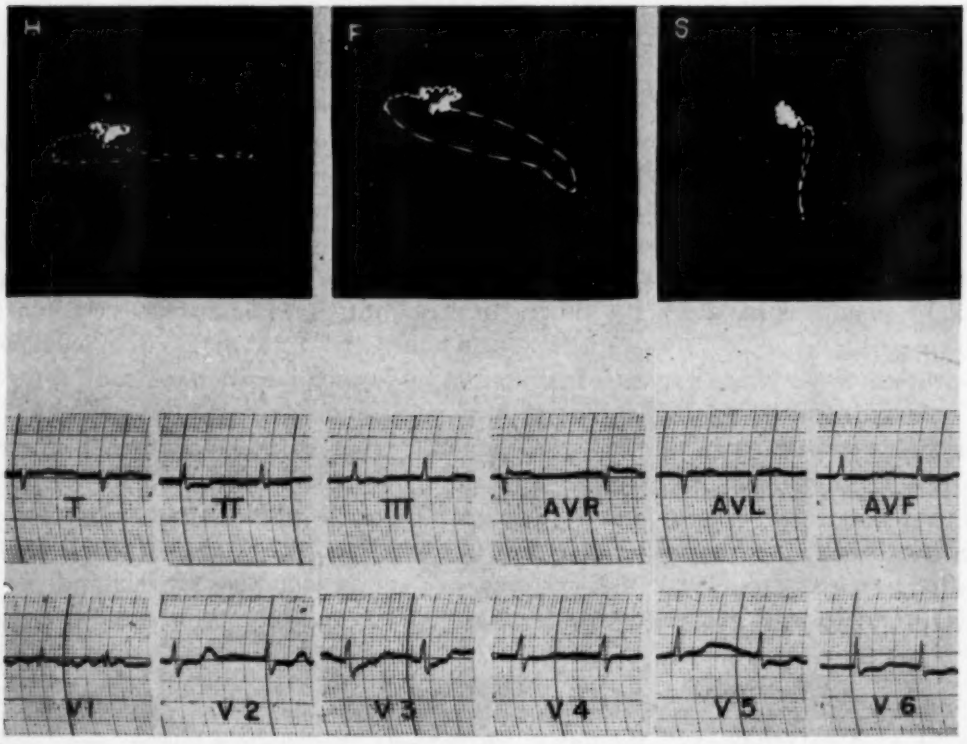

Fig. 1.—A 39-year-old woman with severe mitral stenosis, pulmonary hypertension, and right ventricular failure. The ECG is not diagnostic of RVH, whereas the SVCG shows a characteristic pattern of RVH. (In all figures, *H*, *F*, and *S* represent the horizontal, frontal, and sagittal QRS loops, respectively.)

Primary Heart Disease.—Fig. 1 illustrates the records obtained from a 39-year-old woman with isolated mitral stenosis. The pulmonary arterial pressure was moderately elevated at cardiac catheterization. X-ray and fluoroscopy of the chest showed moderate, generalized cardiac enlargement with a straight left cardiac border and enlargement of the left auricle and right ventricle. At surgery this patient showed a very small mitral opening without evidence of a regurgitant jet. The SVCG shows a typical pattern of RVH with reversal of the usual direction of inscription in the horizontal and sagittal planes, and with anterior displacement of the loops. The ECG is not diagnostic of RVH, although there is some prominence of the R waves from the right precordium.

TABLE I

1° HEART DISEASE	TOTAL NUMBER OF CASES	NUMBER SHOWING RVH CLINICALLY AND/OR PATHOLOGICALLY	NUMBER SHOWING RVH BY:	
			ECG	SVCG
RHD or congenital heart disease with RVH	8	8	4	8
1° Pulmonary disease without evidence of RVH	14	0	0	0
1° Pulmonary disease with evidence of RVH	5	5	1	3

In this tracing, the SVCG loops are not displaced sufficiently anteriorly, to the right, and superiorly to produce to produce the large R waves in aV_R and V_1 and V_2 that are usually found in RVH. We have seen other SVCG's in which the loops were displaced in a similar manner but to a more marked degree, so that the main portion of the loop extended to the right and posteriorly, and away from the area of derivation of the right precordial leads. In these, the R waves from the right precordium were again not enlarged, although prominent R waves were found in aV_R.

Fig. 2 is from a 30-year-old man with isolated pulmonic stenosis. RVH was apparent by x-ray and fluoroscopy, and a very high ventricular pressure was recorded during cardiac catheteri-

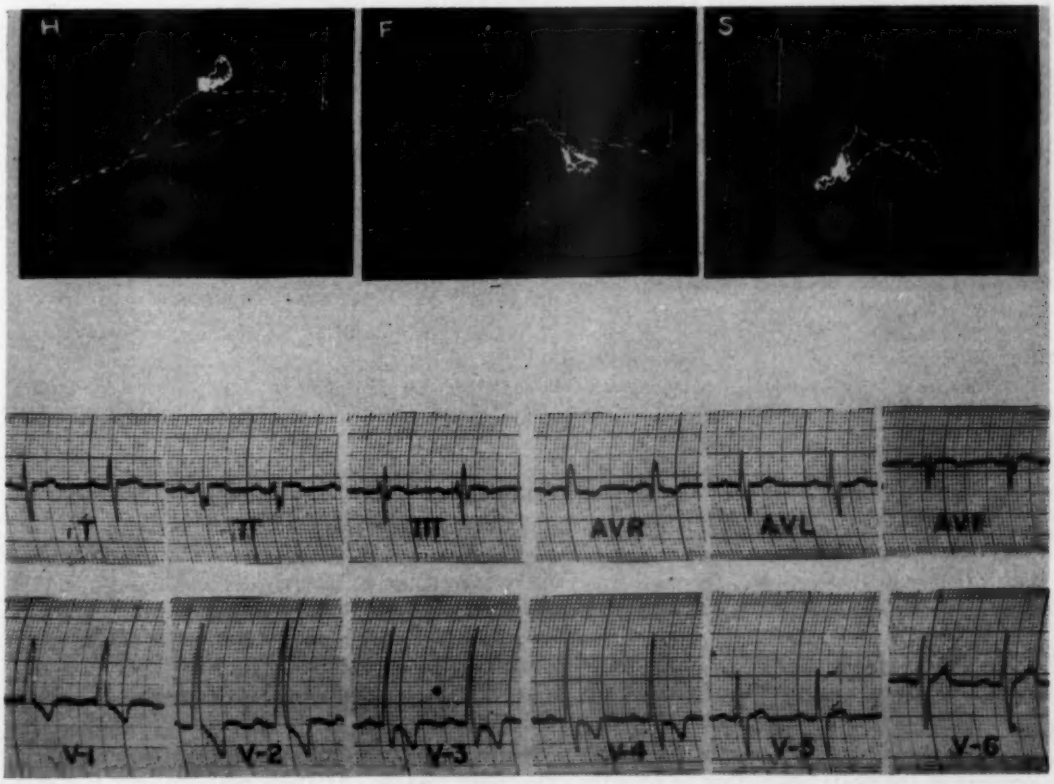

Fig. 2.—A 30-year-old man with isolated pulmonic stenosis, high right ventricular pressure, RVH, and without cardiac failure. Although the ECG suggests RBBB, the SVCG shows a typical pattern of RVH with a terminal conduction delay.

zation. No heart failure was present. The SVCG from the patient was typical of RVH, showing reversal of the normal direction of inscription in the horizontal and sagittal planes and displacement of the loops anteriorly, to the right, and somewhat superiorly. The ECG in this patient reveals very large and broad R waves in aV_R and from the right precordium. Although the usual rsR pattern in V_1 of RBBB is not present in this patient, the wide (.10 second) QRS time suggests the presence of RBBB. The SVCG militates against this interpretation since the characteristic pattern of a long appendage with slow conduction is absent.

Pulmonary Heart Disease.—In patients with slight or moderate pulmonary emphysema and fibrosis (of insufficient degree to give rise to severe symptoms) the SVCG, as well as the ECG's, was found to be unremarkable.

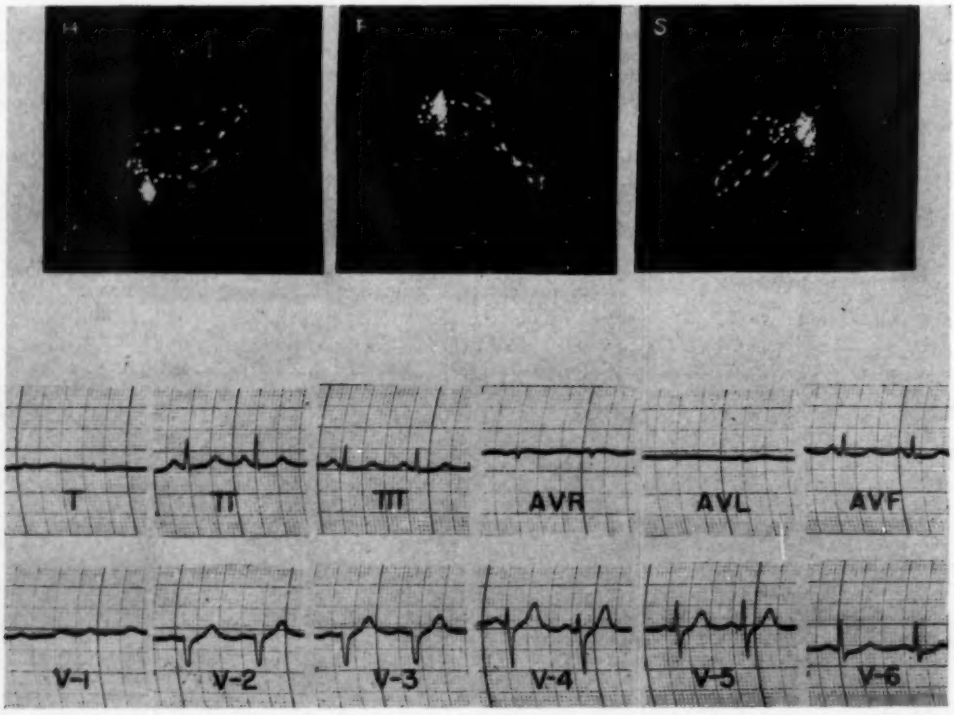

Fig. 3.—A 77-year-old man with severe pulmonary emphysema. The ECG shows vertical cardiac position and clockwise rotation about the long axis. The SVCG shows clockwise rotation about the horizontal axis with displacement of the vector loops posteriorly.

Fig. 3 is representative of one of a number of patients with more marked pulmonary emphysema. In these patients, the emphysema and fibrosis were apparent by x-ray and fluoroscopy. The heart shadows were invariably small in all dimensions, and there was no evidence of right heart failure in these cases. Dyspnea was usually present on mild exertion, and slight cyanosis was often present. There was no polycythemia, and no elevation of the plasma CO_2 content. The SVCG shows a marked clockwise rotation about a vertical axis in the horizontal plane and a posterior displacement of loops in the horizontal and sagittal planes. The direction of inscription in all planes is normal. The ECG shows a vertical position of the heart with clockwise rotation about its long axis.

The SVCG and ECG pattern was felt to be due to the change of electrical and anatomical position of the heart, secondary to primary pulmonary disease. In these patients, the R waves from the right precordium were characteristically small and R aV_R usually so. In one patient (Fig. 4) the R waves were absent in Leads V_1, V_2, and V_3. This change was thought to be due to marked rotation, rather than to an anteroseptal myocardial infarction, though this differentiation may be extremely difficult.

In patients with advanced chronic pulmonary disease, with evidence of right ventricular hypertrophy and failure, one of two patterns was found. The first of these was similar to that found in RVH of primary cardiac origin. The second is similar to that discussed in Figs. 3 and 4 and is illustrated in Fig. 5. The rotational pattern (clockwise in the horizontal loop) is evident, and no evidence of RVH is seen in the SVCG or in the ECG. It is suggested that in this type of tracing, the rotational pattern obscures the conventional pattern of RVH in the right precordial leads, although a prominent R aV_R may be present if the spatial vector loop extends sufficiently far to the right, and superiorly.

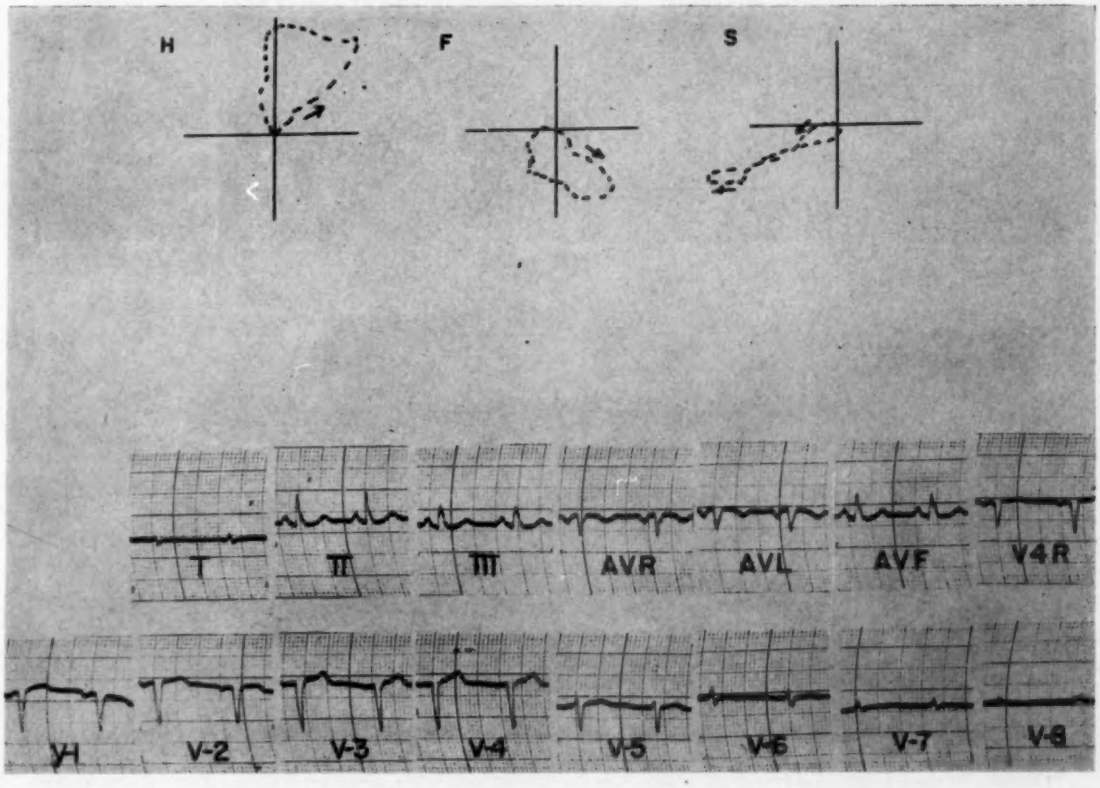

Fig. 4.—A 79-year-old man with marked pulmonary emphysema and slight fibrosis. The ECG shows vertical cardiac position and an absence of R waves over the right precordium, simulating anteroseptal myocardial infarction. The SVCG shows marked clockwise rotation of the vector loops with posterior displacement.

DISCUSSION

The findings in primary heart disease are essentially in accord with those previously reported by other authors.[1-4] The SVCG appears to offer a more sensitive method of diagnosis of RVH than does the ECG. The basic patterns of RVH in congenital heart disease have been pointed out by Lasser and coworkers,[2] and these findings have been extended to patients with mitral stenosis by Elek and associates.[3] The present study extends the interpretation to the diagnosis of right ventricular hypertrophy patterns secondary to chronic pulmo-

nary disease. Assuming the precordial ECG leads to be derivatives of the horizontal plane of the SVCG, the failure of development of the large R wave in the right precordial leads in RVH would appear to be contingent to the spatial orientation of the loops away from their area of derivation. Under this circumstance, the SVCG may show a typical pattern of RVH while the ECG may show equivocal or negative findings relative to RVH. It has also been previously pointed out that the differentiation between RVH and RBBB is facilitated by the SVCG.[1] (This appears to be more particularly true in distinguishing atypical patterns of LVH and LBBB.)

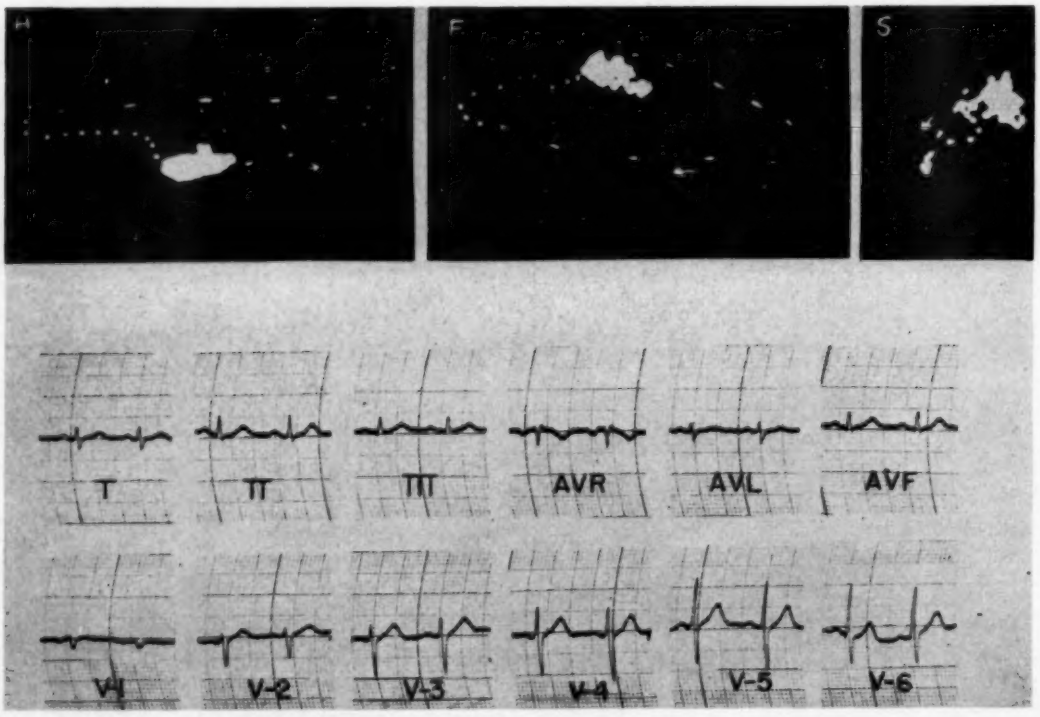

Fig. 5.—A 55-year-old man with severe pulmonary emphysema and fibrosis, RVH, and myocardial insufficiency. The ECG shows vertical cardiac position and clockwise rotation about the long axis. The SVCG shows clockwise rotation about the horizontal axis with posterior displacement of the vector loops.

The study of patients with pulmonary emphysema with and without cor pulmonale is of interest. The posterior rotation of the horizontal loop in pulmonary emphysema has been pointed out by Grishman and Scherlis[1] and is confirmed in this study. While some patients with cor pulmonale show a typical SVCG and ECG pattern of RVH, in others the pattern is that of clockwise rotation in the horizontal plane. In these, RVH is not apparent in either the SVCG or ECG. Some patients with RVH secondary to congenital or rheumatic heart disease also appear to have clockwise rotation in the horizontal plane. This may be evident when the initial component of the SVCG loop in the horizontal plane is directed to the left and posteriorly (normal is to the right and anteriorly) and is offered as an alternative explanation to that of Fowler and

Helm,[4] who have suggested that the septal activation in RVH may be reversed and thus accounts for the qR pattern in the right precordial leads that may occur in RVH. In cases with marked rotation, the absence of the initial deflection of the horizontal loop to the right and anteriorly is reflected as an absence of R waves from the right precordial ECG leads, which can readily be misinterpreted both electrocardiographically and vectorcardiographically as anteroseptal myocardial infarction.

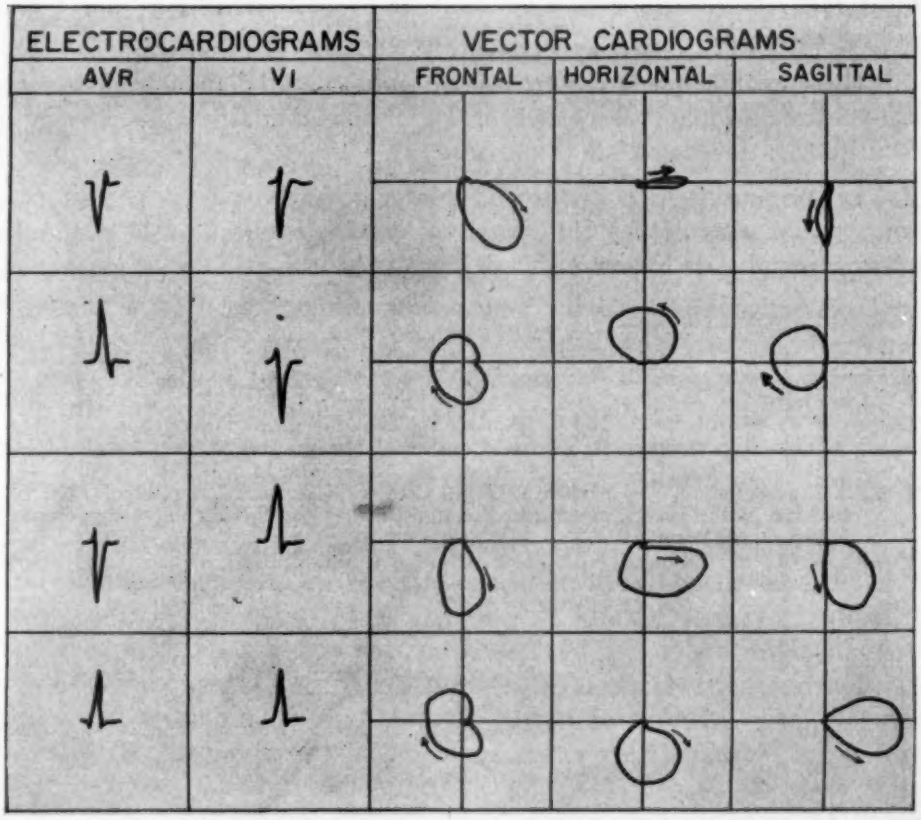

Fig. 6.—Schematic presentation of common type of vector loops associated with various ECG patterns occurring in RVH.

The prominent R in aV_R, which is often found in RVH, appears to be due to the extension of the spatial vector loop to the right, and particularly so when the loop is directed to the right and superiorly. This change should be distinguished from the prominent R in aV_R seen in patients with myocardial infarction in the region of the cardiac apex and is facilitated by the SVCG.

Fig. 6 summarizes schematically the common type of SVCG loops associated with the different patterns of ECG seen in RVH.

SUMMARY

1. SVCG studies have been made in twenty-seven patients with either heart disease and RVH, or with chronic pulmonary disease, and correlated with the ECG and clinical finding.

2. The SVCG may show RVH when the standard ECG shows normal or equivocal finding, and the reasons for the disparity have been discussed.

3. The presence of pulmonary emphysema introduces rotational changes in the SVCG which tend to obscure the ECG pattern of RVH.

SUMMARIO IN INTERLINGUA

1. Esseva executate studios spatio-vectocardiographic, correlationate con constatationes electrocardiographic e clinic, in 27 patientes con o morbo cardiac e hypertrophia dextero-ventricular o chronic morbo pulmonar.

2. Il es possibile que le vectorcardiogramma spatial demonstra hypertrophia dextero-ventricular quando le electrocardiogramma standard es normal o equivoc. Es discutite le rationes pro iste disparitate.

3. Le presentia de emphysema pulmonar introduce in le vectorcardiogramma spatial alterationes rotational que tende a obscurar le configuration electrocardiographic de hypertrophia dextero-ventricular.

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OBSERVATIONS ON THE RELATIONS OF ELECTRICAL AND MECHANICAL EVENTS IN CARDIAC ARRYTHMIAS

M. ELIAKIM, M.D., AND K. BRAUN, M.D.

JERUSALEM, ISRAEL

THE physiology of ectopic premature beats has been studied extensively in the dog by Wiggers.[1] He found that in premature beats the short initial length of the muscle fibers and the small presystolic blood volume resulted in reduction of the systolic pressure and in shortening of the ejection phase. The aberrant path of excitation led to reduced systolic pressure but prolonged the ejection phase, thus partially counteracting the effect on the small presystolic blood volume. Wiggers did not comment on the variations in the period between the beginning of the electrical and of mechanical ventricular systoles of premature beats.

The introduction of catheterization of the right heart in human beings has made it possible to study directly the dynamics of the heartbeat and to correlate the electrical and mechanical events accompanying normal and ectopic beats. The period between the beginning of the Q wave in the electrocardiogram and the rise in right ventricular pressure (the electrical-mechanical latent period) in man, was first examined by Richards and associates[2] and later by Coblentz and associates.[3]

These workers reported a latent period of 0.075 ± 0.04 second during normal beats and 0.074 second in premature beats. However, Testoni and associates[4] using an oscillographic method,[5] found the period between the beginning of the Q wave and the beginning of the intraventricular oscillations in healthy individuals to be 0.048 (0.04 to 0.08) second.

In the following report we present observations on the course of the electrical and mechanical events accompanying ectopic beats and other cardiac arrhythmias. The material was selected with a view to obtaining additional information on factors influencing the mechanical response of the right ventricular myocardium in disturbances of rhythm. Particular attention was paid to the length of the electrical-mechanical latent period.

MATERIAL AND METHODS

The electrocardiogram and right ventricular pressure were recorded simultaneously by a Sanborn twin-channel direct recorder during cardiac catheterization of 22 patients. Of these, 16 suffered from mitral stenosis due to rheu-

From the Cardiovascular Laboratory and Department of Internal Medicine, Division 'B', Rothschild Hadassah University Hospital.

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matic heart disease, 5 from congenital heart disease, and one from cor pulmonale. Two patients had auricular fibrillation. In 20 patients ectopic beats appeared, usually, when the tip of the catheter was in the right ventricle. In 17 of these, ectopic premature beats of ventricular and/or supraventricular origin were recorded. Fusion beats, mechanical alternans, and shifting pacemaker with aberrant ventricular conduction were registered in the remaining three cases, respectively.

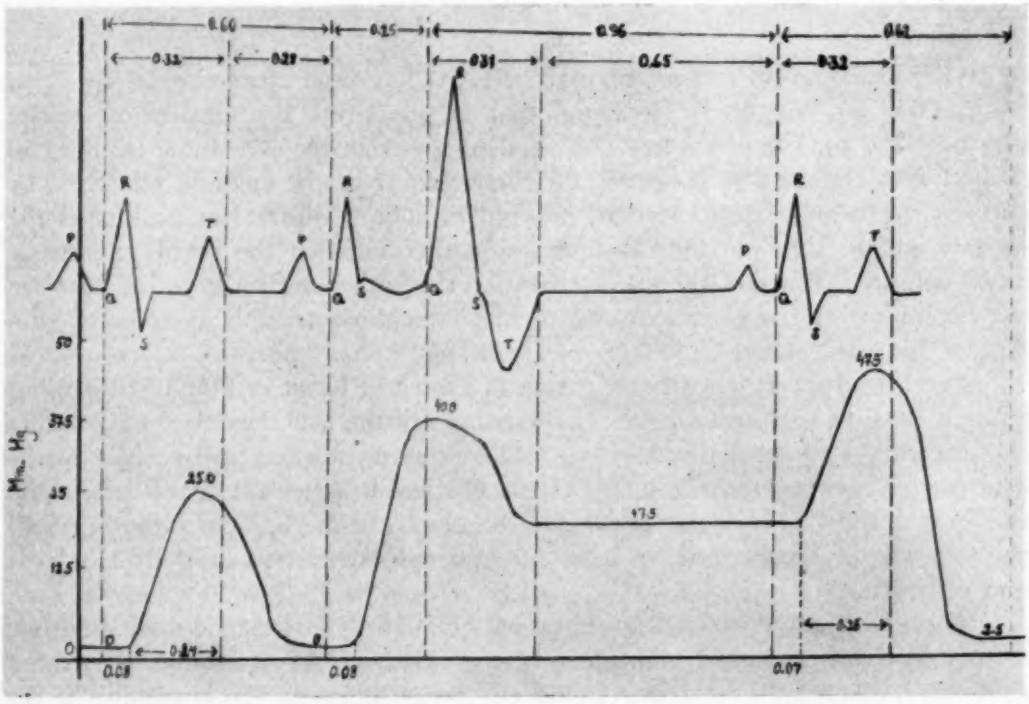

Fig. 1.—Ventricular ectopic beat appearing in the ejection phase of the antecedent beat. The ectopic beat causes an apparent broadening of the mechanical systole of the previous normal beat and maintains its diastolic pressure high throughout the long compensatory pause. The higher systolic pressure of the second beat is not due to the ectopic beat but probably to respiratory variations which were noted throughout the whole record. (Case of cor pulmonale.)

The electrical-mechanical latent period (EMLP), the length of the electrical and the mechanical systoles, and the height of the initial, systolic and the end diastolic pressures were measured and compared in normal and abnormal beats. The EMLP was considered slightly prolonged when it exceeded the EMLP of the normal beats by 0.02 second or less, and prolonged when it was at least 0.03 second longer. The systolic pressure was considered slightly decreased when it was less than 10 mm. Hg lower than that of the normal beats, and decreased when it was lower by 10 mm. Hg or more. The time lag due to mechanical transmission from the heart to the recorder was found to be 0.01 second, and was not taken into consideration when measuring the time intervals between the beginning of the electrical and the mechanical events.

The premature beats were classified as occurring in the ejection phase, isometric ventricular relaxation phase, rapid ventricular filling phase, or in the diastase of the preceding beat. The end of the ejection phase was defined by the projection of the end of the T wave on the ventricular pressure curve, while the other three phases were determined by the changes in contour of the ventricular pressure curve according to the criteria of Wiggers.[1] For the sake of clarity the figures presented are enlarged tracings of the originals.

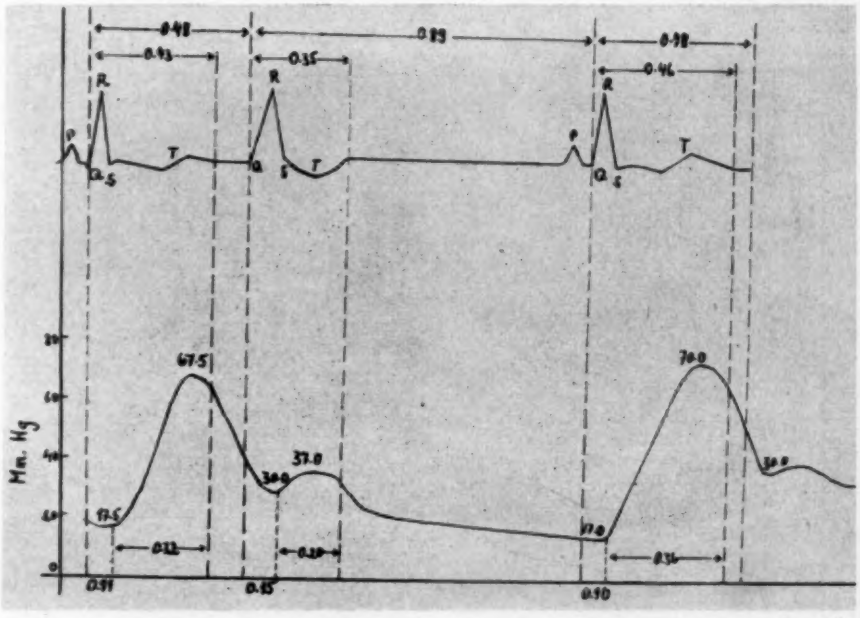

Fig. 2.—Ventricular ectopic beat appearing in the isometric ventricular relaxation phase of the antecedent beat. Note the higher initial tension, longer EMLP, lower systolic pressure, and shorter mechanical systole.

RESULTS

Ectopic Beats.—Ectopic beats appearing during the ejection phase of the antecedent beat (Fig. 1) were recorded three times in one patient. These premature electrical systoles did not result in discrete ventricular contractions (i.e., they were frustrane). They caused, however, broadening of the curve of mechanical systole of the preceding beat and maintained its diastolic pressure at a level higher than that of the normal beats.

Ectopic beats appearing during the isometric ventricular relaxation phase of the antecedent beat (Fig. 2) were recorded 27 times. All but 3 were registered in patients with right ventricular hypertension. One beat was of atrial origin, 19 were of right ventricular, and 7 of left ventricular origin. The atrial premature beat was characterized by a normal EMLP and initial ventricular pressure, and by decreased systolic pressure. Four of the ventricular premature beats did not result in separate ventricular contractions. In the remaining 22 beats the EMLP was prolonged in all, the initial pressure elevated in 20, the

systolic pressure decreased in 20, and the mechanical systole shortened in 15 cases. The origin of the ectopic beat in either the right or left ventricle, as determined from the electrocardiogram, did not seem to influence the length of the EMLP or the height of the systolic pressure.

It is of particular interest that the initial pressure was normal in 7 beats in spite of their appearance in the isometric ventricular relaxation phase. Four of these occurred in patients with normal right ventricular diastolic pressure. The systolic pressure during 6 of these 7 beats was low, while during the seventh it was normal (Fig. 3).

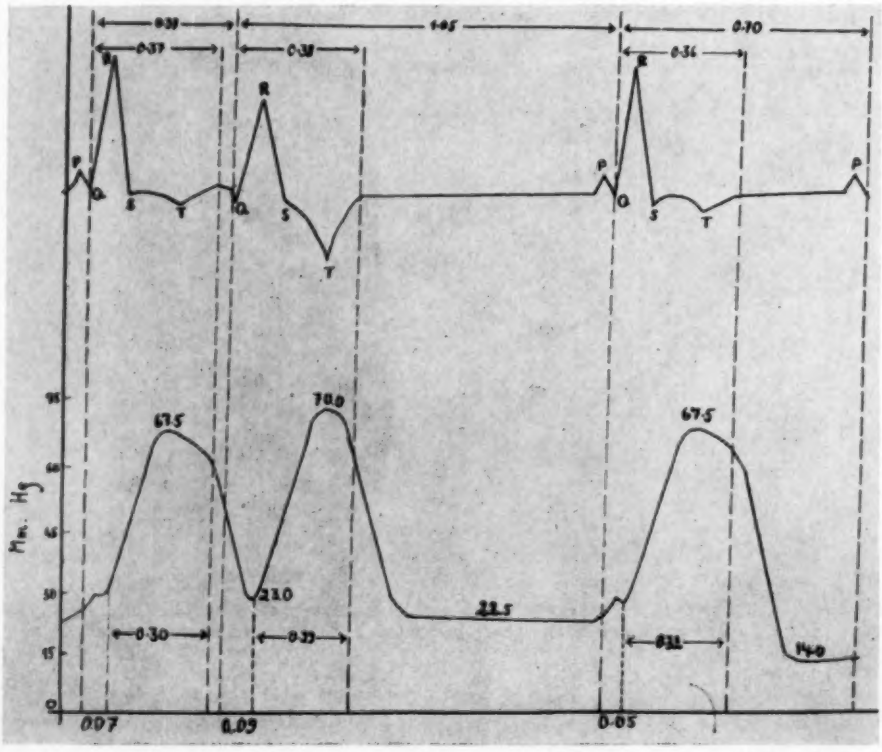

Fig. 3.—Ventricular ectopic beat appearing in the isometric ventricular relaxation phase of the antecedent beat. The systolic pressure is normal although the EMLP is slightly prolonged and the mechanical systole slightly shortened. Note the normal initial pressure.

Ectopic beats appearing during the rapid ventricular filling phase of the antecedent beat (Fig. 4) were recorded 27 times. All but one were registered in patients with right ventricular hypertension. Three beats were of nodal origin, 18 of right ventricular, and 6 were of left ventricular origin. The nodal premature beats were characterized by a normal EMLP and initial pressure, slightly decreased systolic pressure and shortened mechanical systole. In the 24 ventricular beats the EMLP was prolonged in 12 of the cases, the inital pressure elevated in 16, the systolic pressure lowered in 10, and the mechanical systole shortened in 7 of the cases. In 13 cases the mechanical systole was not measurable. Here again the site of origin of the ventricular ectopic beats did not seem to influence the pressure curves.

Ectopic beats appearing during the diastase of the antecedent beat (Fig. 5) were recorded 19 times. All but 2 were registered in patients with right ventricular hypertension. Three beats were of nodal origin, 13 of right ventricular, and 3 were of left ventricular origin. The nodal premature beats were characterized by normal EMLP, normal initial and systolic pressures, and shortened mechanical systole. In the 16 ventricular beats the EMLP was normal or only slightly prolonged in all of the cases, the initial pressure was normal in all, and the systolic pressure normal or only slightly decreased in 12. The mechanical systole was shortened in 3 cases, prolonged in 4, and normal in 4 cases. In 5 of the cases the mechanical systole was not measurable.

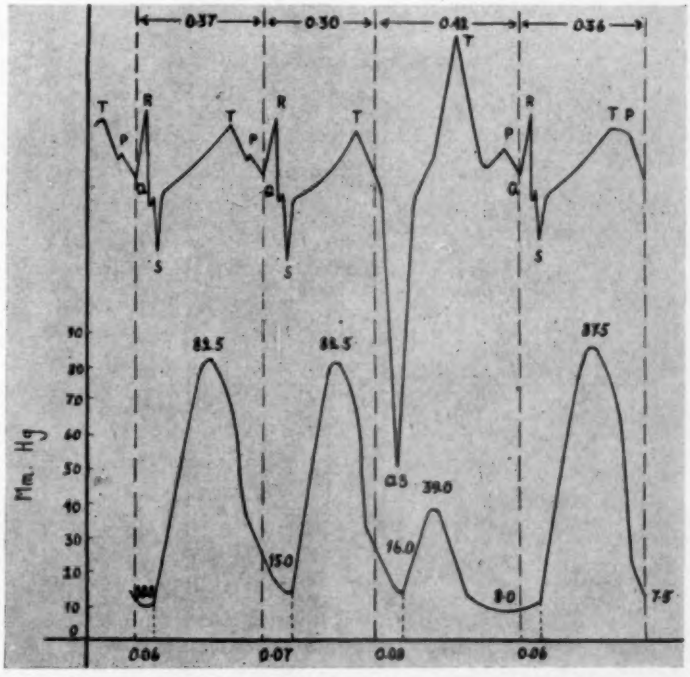

Fig. 4.—Ventricular ectopic beat appearing in the rapid ventricular filling phase of the antecedent beat. The systolic pressure is much lower than normal although the initial pressure and the EMLP are almost normal. The mechanical systole cannot be measured because of fusion of the P and T waves throughout the electrocardiogram.

Fig. 5 illustrates the importance of the time of appearance of the ectopic beats in influencing the EMLP and the systolic pressure. In this record ventricular ectopic beats from one and the same focus, when appearing in different phases of the diastole, had different EMLP's and systolic pressures.

Table I shows that the chance of finding both a normal EMLP and normal systolic pressures increased as the ectopic beat appeared progressively later in the diastole of the antecedent beat.

During a bout of 3 ventricular ectopic beats there was progressive lowering of the systolic pressure and a mild rise of the diastolic pressure (Fig. 6). The first ectopic beat probably appeared in the rapid ventricular filling phase of the preceding normal beat and had an EMLP equal to that of the normal beats.

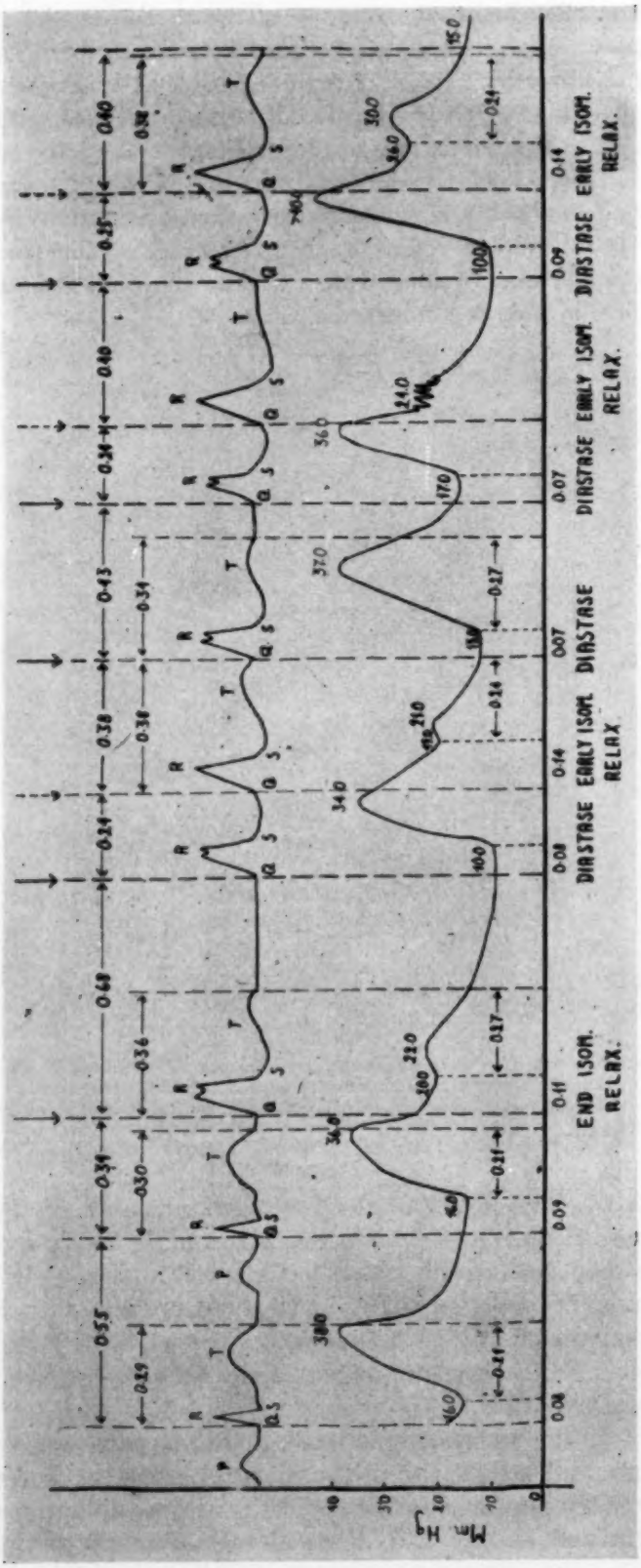

Fig. 5.—Ventricular ectopic beats from two foci. Ectopic beats marked with an unbroken arrow originate from the first focus; all of them but the first in the figure appear in the diastase of the antecedent beat and produce normal ventricular pressure curves. Ectopic beats marked with a broken arrow originate from the second focus and appear in the isometric relaxation phase. They have prolonged EMLP's and cause insignificant rises in pressure. The first ectopic beat from the first focus appears in the isometric relaxation phase too and has the same characteristics as the beats from the second focus. The fourth beat on this record is an escape beat which causes an apparently normal ventricular contraction.

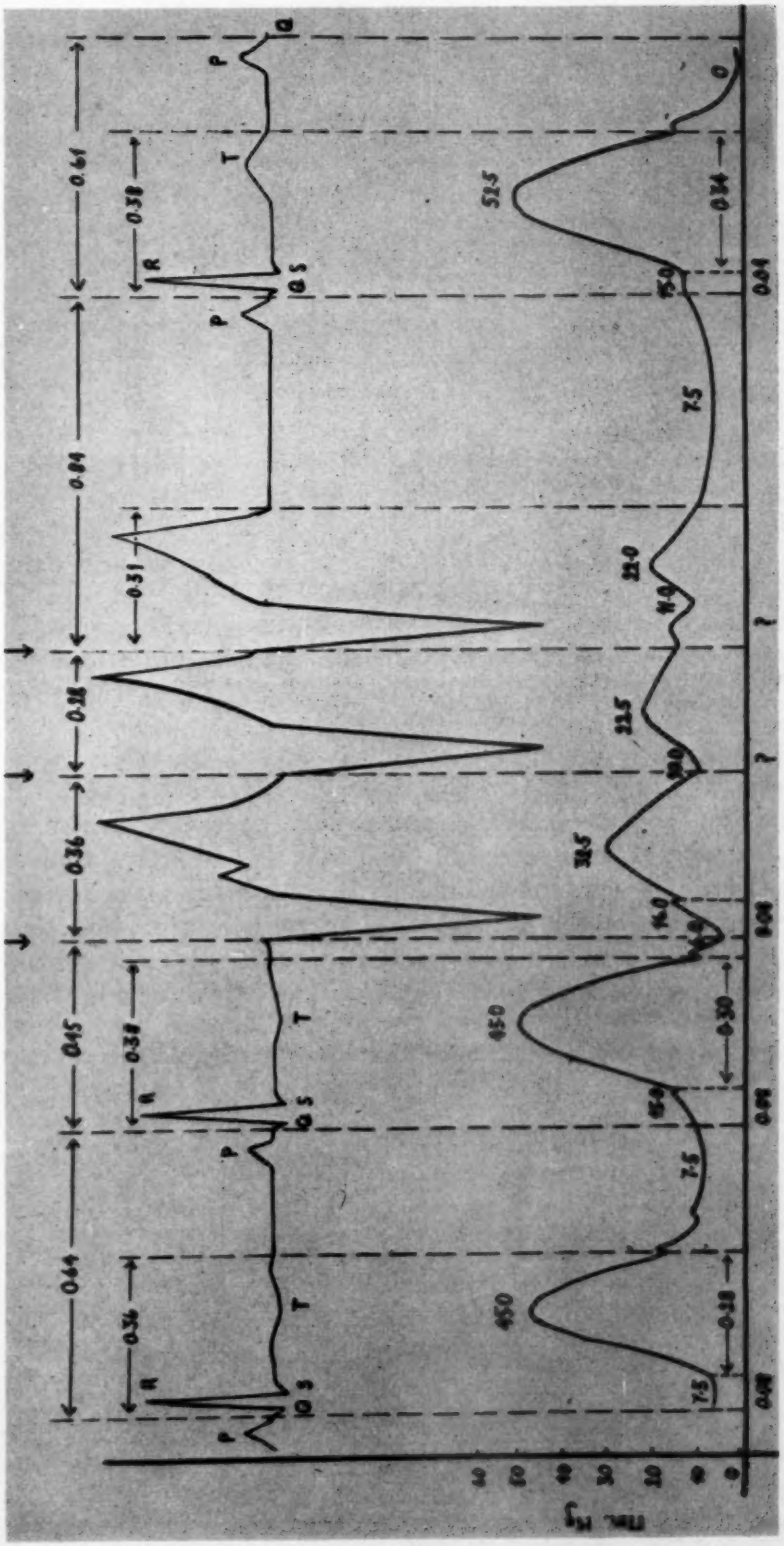

Fig. 6.—Effect of a bout of ventricular ectopic beats on ventricular pressures. Note the progressive decrease in the systolic and the slight rise in the diastolic pressure.

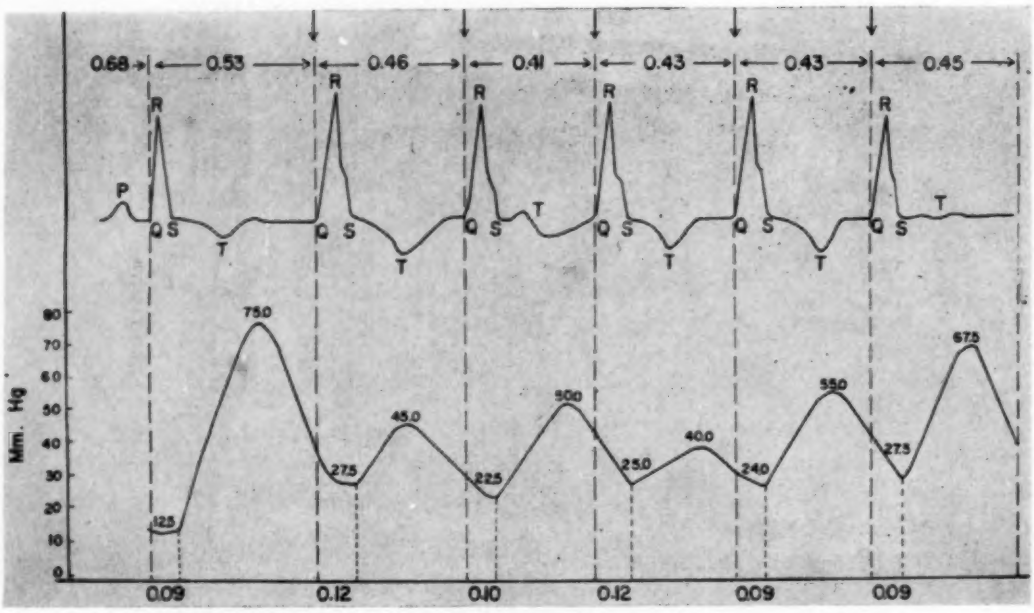

Fig. 7.—Effect of a bout of ventricular ectopic beats on ventricular pressures. The systolic pressure tends to rise and the EMLP to shorten during the course of the bout. Note that beats with a prolonged EMLP have a low systolic pressure, and vice versa.

In the 2 subsequent ectopic beats it was difficult to measure the EMLP accurately. In the first postectopic beat not only was the systolic pressure higher and the EMLP shorter than normal, but the T wave was upright, as opposed to the almost isoelectric T waves of the normal beats preceding the ectopic ones.

On the other hand, in one patient a run of ventricular ectopic beats was recorded which was characterized by a low but progressively rising systolic pressure and an elevated diastolic pressure (Fig. 7). The first ectopic beat appeared

TABLE I. EFFECT OF TIME OF APPEARANCE OF THE ECTOPIC BEAT ON THE ELECTRICAL-MECHANICAL LATENT PERIOD AND SYSTOLIC PRESSURE

PHASE OF APPEARANCE	TOTAL NUMBER OF BEATS	SYSTOLIC PRESSURE NORMAL			SYSTOLIC PRESSURE DECREASED			FRUSTRANE
		EMLP NORMAL	EMLP SLIGHTLY PROLONGED	EMLP PROLONGED	EMLP NORMAL	EMLP SLIGHTLY PROLONGED	EMLP PROLONGED	
Ejection	3	—	—	—	—	—	—	3
Isometric Ventricular Relaxation	27	0	0	2	1	0	20	4
Rapid Ventricular Filling	27	1	1	4	9*	0	12†	0
Diastase	19	10	1	3	2	3‡	0	0
Totals	76	11	2	9	12	3	32	7

*In 4 cases systolic pressure only slightly decreased.
†In 7 cases systolic pressure only slightly decreased.
‡In 2 cases systolic pressure only slightly decreased.

in the isometric ventricular relaxation phase of the preceding beat and had a markedly prolonged EMLP. In the subsequent beats there was an inverse relationship between the length of the EMLP and the height of the systolic pressure.

In a case of bigeminy all the premature beats apparently came from the same ectopic focus in the ventricle and appeared in the rapid ventricular filling phase of the preceding beat (Fig. 8). They were characterized by a prolonged EMLP, high initial pressure, and slightly decreased systolic pressure.

Ventricular Fusion Beats.—Fusion beats, in which the ventricular premature contraction occurred immediately after the auricular systole (P wave) were recorded from a case with tetralogy of Fallot (Fig. 9). During normal beats the electrocardiogram showed giant P waves and tiny QRS complexes in the standard Leads II and III. The fusion beats where characterized by normal P waves, shortening of the P-Q interval, broadening of the QRS complexes, shortening of the P-J (P-Q + QRS) interval, and peaked T waves. The EMLP of the fusion beats was prolonged and the systolic pressure markedly decreased.

Mechanical Alternans.—Mechanical alternans, not related to an irregularly varying amplitude of the ventricular complexes in the electrocardiogram, was recorded immediately before operation in a case of pulmonary valvular stenosis (Fig. 10). The ventricular rate was about 109 per minute and the Q-Q intervals varied, those preceding the beats with low pressure being slightly shorter. The systolic ventricular pressure was 100 mm. Hg for the "large" beats and 87.5 mm. Hg for the "small" beats. Both low and high pressure beats occurred apparently in the isometric ventricular relaxation phase of the preceding beat, the former earlier than the latter. The small beats had a slightly longer EMLP and higher initial pressure than the large beats.

Immediately following valvotomy the ventricular rate dropped to about 100 per minute but the Q-Q intervals varied in the same manner as before operation. The systolic pressure of the large beats dropped to 75 mm. Hg and of the small beats to 72.5 mm. Hg, so that mechanical alternans almost disappeared. Both small and large beats appeared later in the diastole of the preceding beat as compared with the preoperative record. The mechanical and the electrical systoles became longer and the EMLP of all beats became shorter and equal.

Wandering Pacemaker With Aberrant Ventricular Conduction.—Wandering pacemaker with aberrant ventricular conduction appear in the record shown in Fig. 11. Starting from the third beat in the figure, the P-Q intervals became progressively shorter and the P waves smaller. The Q-Q intervals between the ectopic beats were longer than those between the normal ones because of gradual prolongation of diastole. There was slight prolongation of the EMLP of the abnormal beats, but no change in pressure except for the usual respiratory variations. The ventricular complexes of the abnormal beats were completely different from the normal and the electrical and mechanical systoles of these beats were slightly shortened.

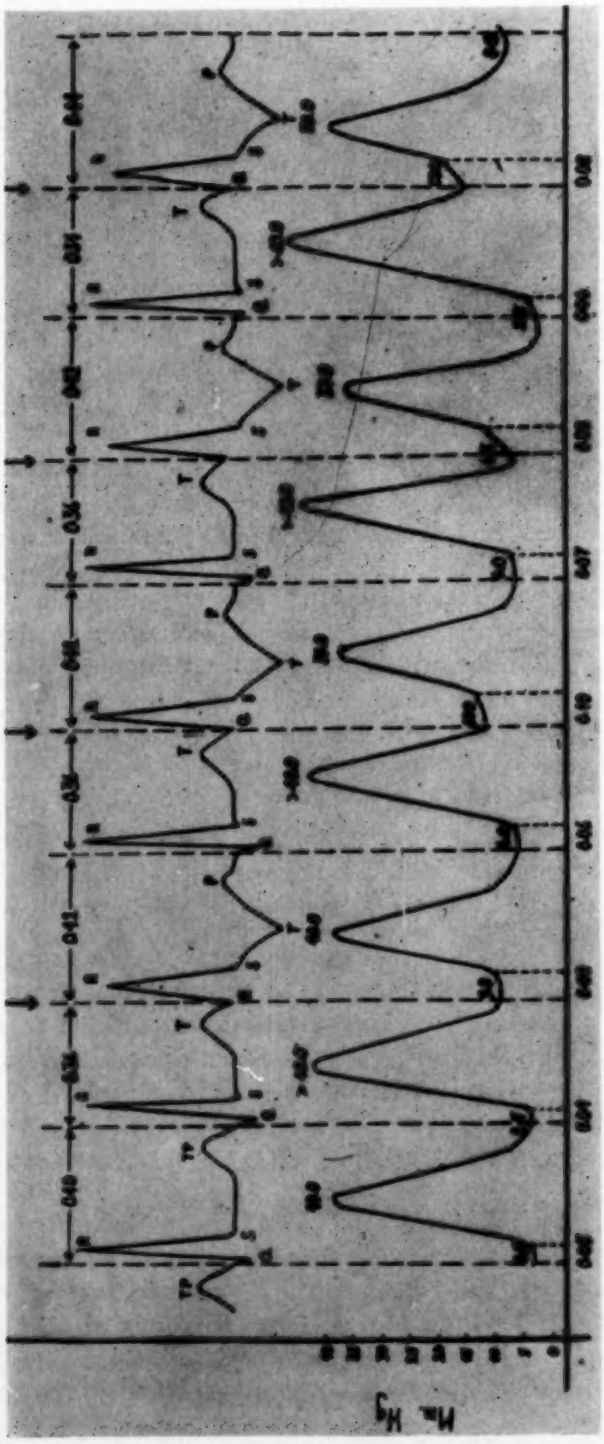

Fig. 8.—Effect of bigeminy on ventricular pressures and on the EMLP. Note the high initial pressure, decreased systolic pressure, and prolonged EMLP of the ectopic beats as compared with the normal beats. The length of the electrical and the mechanical systoles could not be measured because of fusion of the P and T waves.

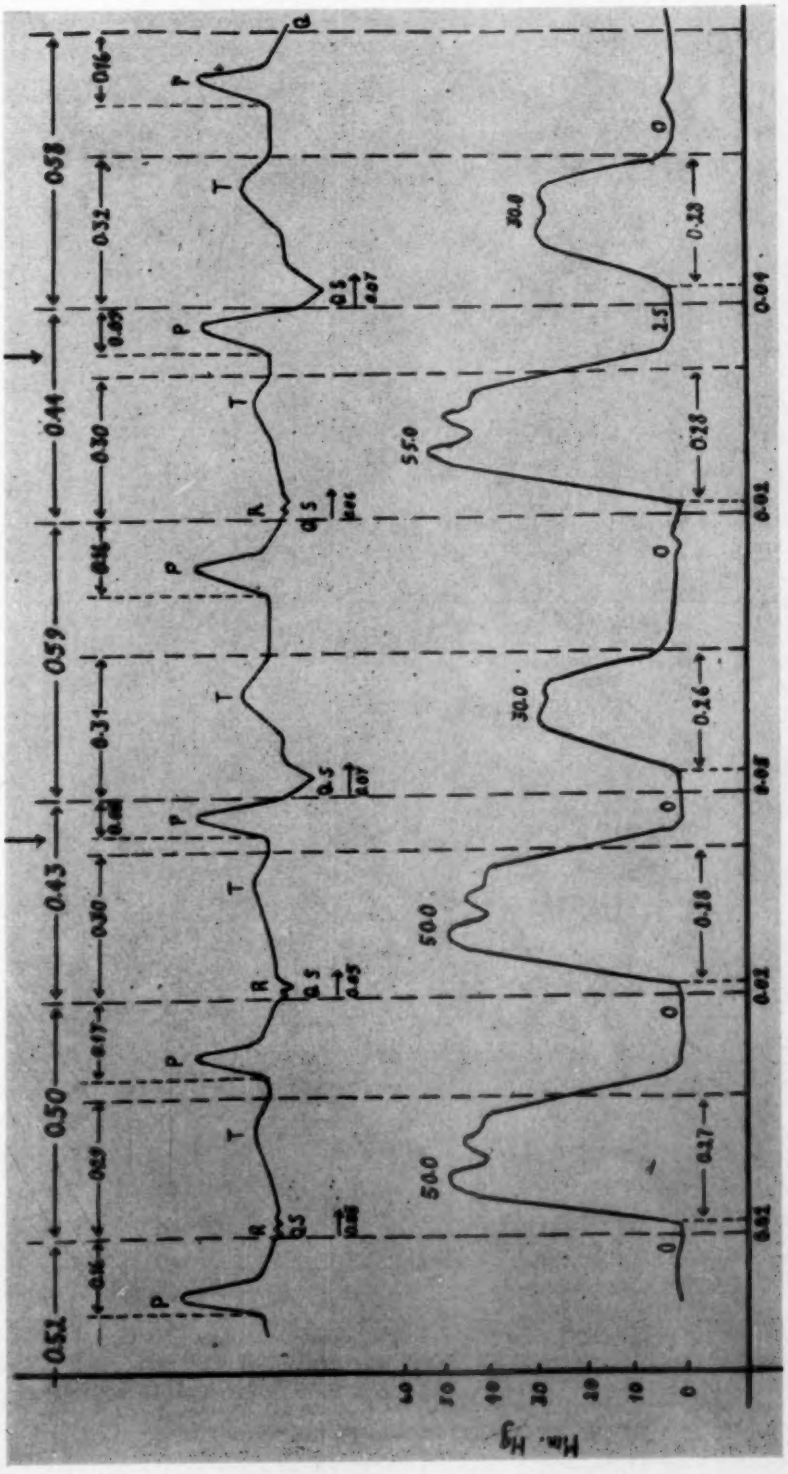

Fig. 9.—Ventricular fusion beats. The normal beats have giant P waves and tiny splintered ventricular complexes. The abnormal beats are characterized by normal P waves and widened ventricular complexes which appear prematurely immediately after the end of the P waves: their EMLP's are prolonged and the systolic pressures lower than those of the normal beats.

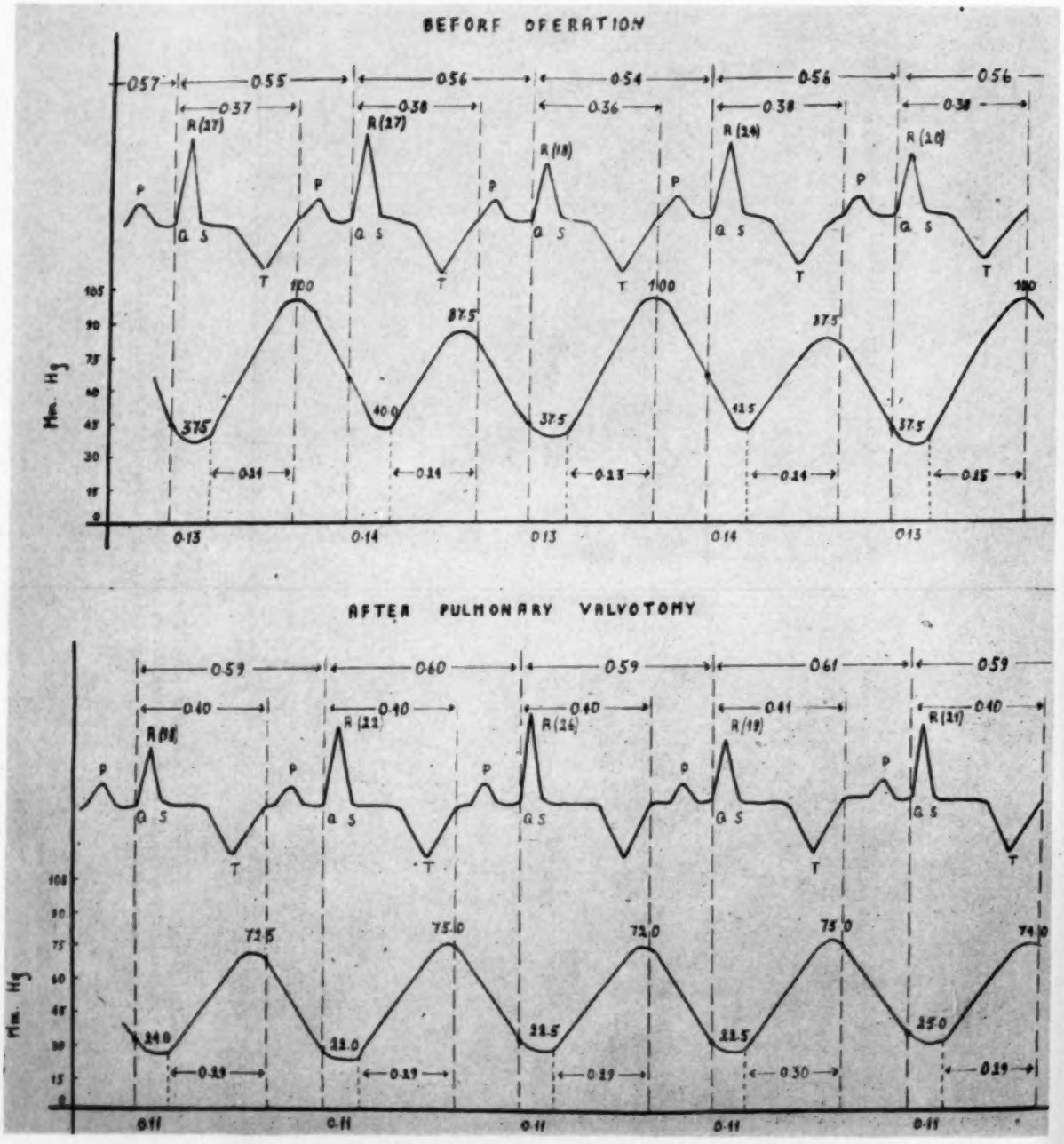

Fig. 10.—Ventricular alternans before and after pulmonary valvotomy. Before operation, the small beats are comparatively premature as seen from the projection of the beginning of the QRS complexes on the ventricular pressure curve. After valvotomy, alternans disappeared almost completely. Note the postoperative drop in ventricular pressure, the slowing of the pulse rate, the shortening and equalizing of the EMLP for all beats, and the prolongation of the electrical and the mechanial systole. (Further details in text.)

Auricular Fibrillation.—In two cases of auricular fibrillation the beats following long Q-Q intervals had low initial and high systolic pressures while beats following short Q-Q intervals were characterized by high initial and low systolic pressures (Fig. 12). After long Q-Q intervals the EMLP tended to be shorter, and vice versa.

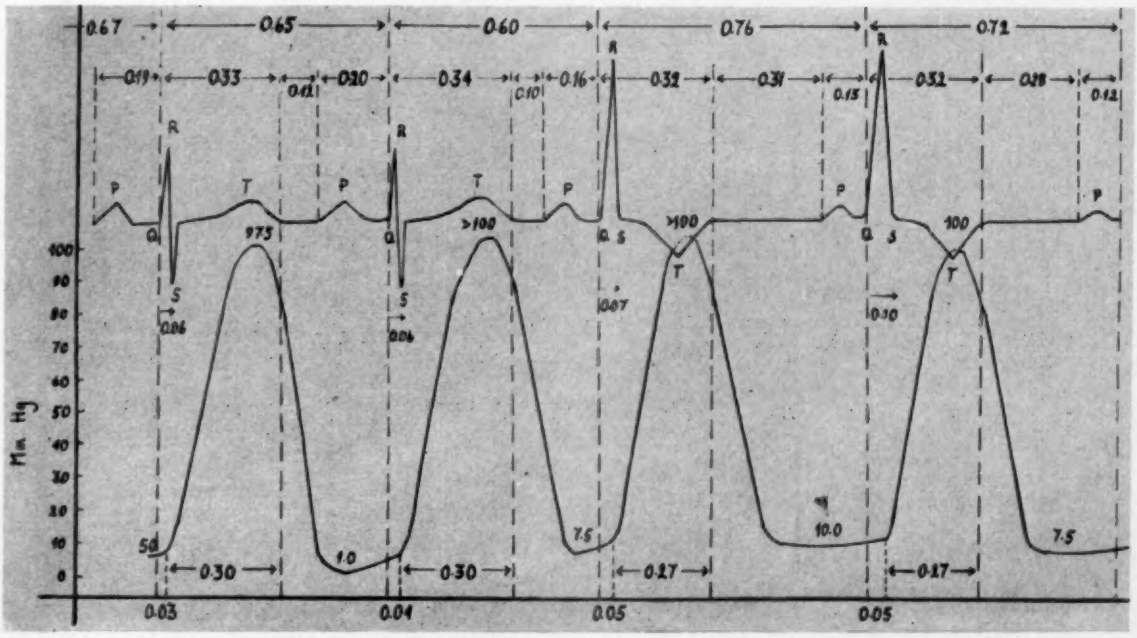

Fig. 11.—Wandering pacemaker with ventricular aberration. The systolic pressures of the pathologic beats are normal but the EMLP's are slightly prolonged.

COMMENT

The data obtained in this study indicate that the length of the EMLP is related to the degree of prematurity of the ectopic beat. The EMLP lengthens as the ectopic beat appears earlier in the diastole of the preceding beat. Therefore even when different ectopic beats originate from the same focus (as far as can be determined from the electrocardiogram) their EMLP's and the induced systolic pressures may vary depending on the time of appearance of the beat (Fig. 5). The probable explanation of this phenomenon is that the earlier the ectopic beat reaches the myocardium, the more refractory the muscle is and, therefore, the longer the time from onset of the electrical stimulus until the rise of the ventricular pressure. Our findings indicate that the conclusion of Richards and associates[2] that the EMLP is normal in most ventricular premature beats is true only when they occur late in the diastole.

Premature beats with prolonged EMLP's generally had lower systolic pressures than normal beats. However, this does not imply a causative relationship between the length of the EMLP and the height of the systolic pressure, since

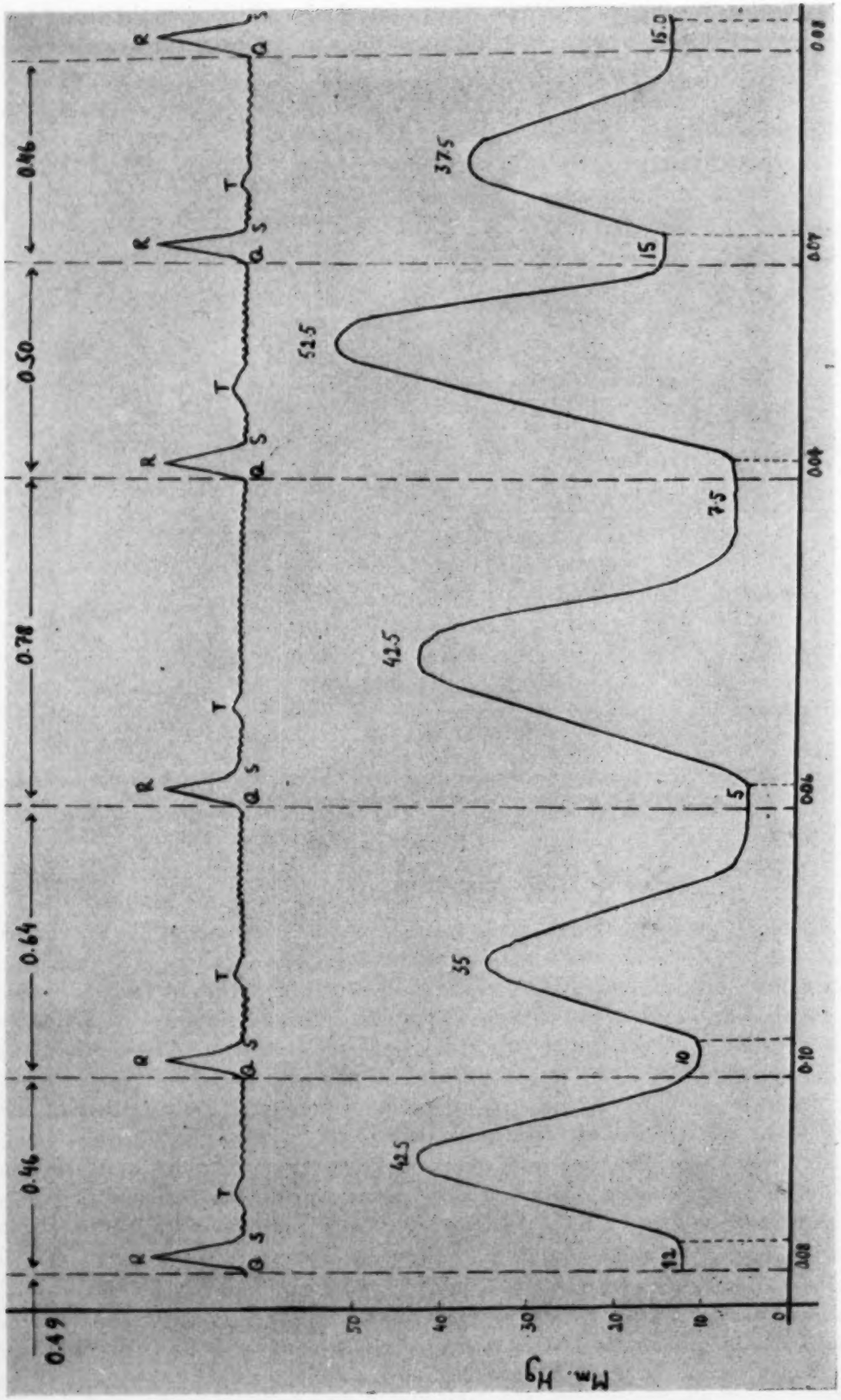

Fig. 12.—Auricular fibrillation. Note the low initial and high systolic pressures in the beats following long cardiac cycles and vice versa. There is an apparent inverse relationship between the length of the EMLP and the length of the previous cardiac cycle.

beats with a prolonged EMLP and a normal systolic pressure, as well as beats with a normal EMLP and a decreased systolic pressure were observed. The appearance of the ectopic beat early in diastole was apparently the cause both of the lengthening of the EMLP and the lowering of the systolic pressure.

The prolongation of the EMLP's of premature beats with normal systolic pressures was probably due to longer duration of spread of the electrical stimulus through the refractory muscle and/or over the aberrant pathway. A similar mechanism may be present in the escape beats (Fig. 5) and in the case of shifting pacemaker with aberrant conduction (Fig. 11). Despite the normal systolic pressures of these beats, their mechanical efficiency was impaired, since the stroke volume was usually decreased as indicated by the shortened mechanical systole.

The finding of normal EMLP in the case of premature beats with decreased systolic pressures may be due to spreading of the electrical stimulus over a pathway very close to the normal or even to short circuiting. The mechanical efficiency of these beats, nevertheless, was diminished because the muscular response to impulses traveling over the short circuit may be weak and because of the small presystolic blood volume.

Our results are in agreement with the findings of other authors[6] that the systolic pressure of the premature beat is, generally, directly related to the length of the preceding diastole. Exceptions occurred in the case of premature beats appearing in the isometric ventricular relaxation phase and giving rise to normal systolic pressures. The majority of these beats were mechanically inefficient, having high initial and small pulse pressures. One beat was found which, despite its early appearance, had a normal initial and a normal pulse pressure, though the mechanical systole was shortened (Fig. 3).

The height of the systolic pressure and the length of the EMLP of the premature beats, therefore, seem to be governed mainly by two factors, the degree of prematurity and the aberrant path of the electrical stimulus.

In pulsus alternans, as opposed to bigeminy, the interval between large and small beats is usually slightly longer than that between small and large beats, though occasionally they may be equal.[7] In our case (Fig. 10) the cycle length between large and small beats, if anything, was slightly shorter than that between small and large beats, thus stimulating bigeminy of sinus origin. The only apparent reason for the decreased systolic pressure and slightly prolonged EMLP of the small beats was their comparative prematurity. This difference in cycle length may account for alternating variations in recovery and conductivity of the myocardium.[8] Prolonged EMLP of the small beats in cases of alternans recorded in the femoral artery was reported by Richards and associates.[2] The almost complete disappearance of alternans immediately after pulmonary valvotomy in our case, was probably caused by improvement of myocardial function accompanying the reduction of right ventricular pressure and by the slowing of the pulse rate, causing the previously small beats to appear later in diastole. This is in agreement with Straub's observation[9] that alternans develops when the heart rate is fast, or the pressure curve so broad that the pressure does not reach normal before the next ventricular excitation supervenes.[9]

In auricular fibrillation an apparent inverse relationship between the length of the EMLP and both the systolic pressure and the length of the preceding cycle was found. This relationship was also noted in direct records of brachial artery pressures.[10] These observations are in contradiction to the findings of Coblentz and associates[3] who did not observe a constant relationship between the cycle length and the EMLP in right ventricular pressure recordings. Messer and co-workers[11] investigated the effect of the cycle length on the time of occurrence of the first heart sound in auricular fibrillation. They found that the first part of the first heart sound in the phonocardiogram was delayed with respect to the Q wave when the preceding cycle was short, and vice versa. These findings are in agreement with ours since the first part of the first heart sound coincides exactly with the onset of the upward deflection of the ventricular pressure curve.[12] Shortening of the EMLP was also observed in normal beats following premature ones when the compensatory pause was long (Fig. 6). This shortening may be due to the quick response of muscle which is less refractory after a long recovery phase.

SUMMARY

1. The latent period between the onset of the electrical and mechanical systoles, the length of the electrical and mechanical systoles and the height of the systolic and diastolic pressures were measured by simultaneous recording of right ventricular pressure and electrocardiogram in twenty-two patients suffering from congential or acquired heart disease, and compared in normal and abnormal beats.

2. Ectopic beats appearing in the ejection phase caused a broadening of the mechanical systole of the antecedent beat and maintained its diastolic pressure at a level higher than normal.

3. Ectopic beats appearing in the isometric ventricular relaxation phase usually had a high initial pressure, prolonged electrical-mechanical latent period, decreased systolic pressure, and shortened mechanical systole.

4. Ectopic beats appearing in the rapid ventricular filling phase had a normal or elevated initial pressure, normal or prolonged electrical-mechanical latent period, normal or decreased systolic pressure and, frequently, shortened mechanical systole.

5. Ectopic beats appearing in the diastase of the antecedent beat had a normal initial pressure, usually a normal electrical-mechanical latent period and systolic pressure, and a short, normal, or prolonged mechanical systole.

6. Successive ectopic beats produced a lowering of systolic pressure and a rise in diastolic pressure in the ventricle, thus interfering with heart performance.

7. Data on cases with ventricular alternans, fusion beats, wandering pacemaker with aberrant ventricular conduction, and auricular fibrillation are presented and discussed.

SUMMARIO IN INTERLINGUA

1. In 22 patientes con congenite o acquirite morbo cardiac, determinationes del pression dextero-ventricular con simultanee registrationes electrocardio-

graphic esseva usate pro mesurar le latente periodo inter le declaration del systoles electric e mechanic, le longor del systoles electric e mechanic, e le elevation del pressiones systolic e diastolic. Le datos esseva comparate pro pulsos normal e anormal.

2. Pulsos ectopic occurrente in le phase de ejection causava un allargamento del systole mechanic del pulso precedente e manteneva le pression diastolic de illo a un nivello supranormal.

3. Pulsos ectopic occurrente in le phase isometric de relaxation ventricular esseva usualmente characterisate per alte pression initial, prolongate periodos atente electrico-mechanic, reducite pression systolic, e un abbreviate systole mechanic.

4. Pulsos ectopic occurrente in le phase de replenamento ventricular rapide habeva un normal o elevate pression initial, un normal o prolongate periodo latente electrico-mechanic, normal o reducite pression systolic, e frequentemente un abbreviate systole mechanic.

5. Pulsos ectopic occurrente in le diastase del pulso precedente habeva un normal pression initial, usualmente un normal periodo latente electrico-mechanic e normal pression systolic, e un breve o normal o prolongate systole mechanic.

6. Successive pulsos ectopic produceva un abassamento del pression systolic e un altiamento del pression diastolic in le ventriculo. Assi illos interfereva con le functionamento del corde.

7. Es presentate e discutite datos ab casos de pulso alternante ventricular, de pulsos fusional, de pacemaker divagante con aberrante conduction ventricular, e de fibrillation auricular.

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MYOCARDIAL INFARCTION COMPLICATED BY LEFT BUNDLE BRANCH BLOCK

STUDIES ON THE MECHANISM OF VENTRICULAR ACTIVITY. XI.

REXFORD KENNAMER, M.D., AND MYRON PRINZMETAL, M.D.

LOS ANGELES, CALIF.

THE coronary Q wave characteristically recorded over chronic left ventricular infarcts has been attributed to transmission of negative cavity potentials through dead muscle to the epicardial surface.[1] Wilson and his associates observed both in experimental animals[2] and in man[3] that such Q waves fail to occur when the infarction is complicated by left bundle branch block. The absence of the coronary Q wave has led to the belief that it is extremely difficult and in many cases impossible to diagnose myocardial infarction in the presence of left bundle branch block. The present study was carried out to evaluate this hypothesis and to detect any possible aids that might be of value in diagnosing the combination of myocardial infarction and bundle branch block.

A. QRS COMPLEX

The experimental study on the change in the QRS complex was performed by cutting the left branch of the bundle of His in nine dogs from four days to four weeks after ligation of the anterior descending artery. The operative techniques, electrocardiographic equipment, and recording methods used in the experiments have been described previously.[4]

Histologic examination revealed three different pathologic patterns in different infarcts or in different parts of the same infarct: (a) fibrosis or necrosis involving the subendocardial region but not affecting the outermost layers of the wall; (b) transmural "holes" or "windows" composed entirely of dead tissue extending from endocardium to epicardium; and (c) patchy lesions involving all levels of the wall but containing variable amounts of viable muscle.[5] Regions of purely subendocardial muscle death generally appeared at the periphery of transmural infarcts. "Holes" of dead tissues were most frequently found near the center of the infarct. Regions of patchy infarction were observed in almost every ventricle, whether or not regions of subendocardial or through-and-through infarction were found in adjacent parts of the ventricle. The

From the Institute for Medical Research, Cedars of Lebanon Hospital, and the Department of Medicine, University of California School of Medicine, Los Angeles, Calif.

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epicardial potentials associated with each of these three types of infarction in ventricles with bundle branch block and their probable relation to precordial leads constitute the subject of the present paper.

As described in a previous report,[6] abnormally large positive deflections are obtained throughout the wall of uninfarcted ventricles with bundle branch block. The magnitude of the R wave is greatest in epicardial leads, decreases as deeper intramural levels are explored, and is smallest in tracings from the endocardial surface. Leads from within the subjacent cavity exhibit pure R waves or Rs waves when the corresponding bundle branch is completely blocked; if a large negative component follows the cavity R wave, the block is incomplete (Fig. 1). These observations are essential to proper interpretation of the following findings in infarcted ventricles with bundle branch block.

RESULTS

(a) *Subendocardial Infarction.*—The intact epicardial surface overlying subendocardial infarcts of the left ventricle yielded essentially normal R or Rs waves prior to the production of left bundle branch block. When the left bundle branch was severed, the surface R wave became abnormally tall, broad, and notched (Fig. 2). This complex was indistinguishable from the large R wave characteristically obtained over uninfarcted left ventricles with bundle branch block (compare Figs. 1 and 2). As in uncomplicated bundle branch block, the epicardial R wave registered over the subendocardial infarct was considerably larger than the positive deflection in simultaneous intracavity leads.

(b) *Through-and-Through Infarctions.*—Prior to the production of bundle branch block, leads from the epicardial surface over through-and-through infarcts presented coronary QS waves essentially identical with the QS deflection recorded simultaneously from the underlying cavity. After the left bundle branch was cut, the surface and cavity tracings again were identical. Pure R or Rs waves were now recorded from the epicardial surface directly over transmural lesions of the ventricle with bundle branch block (Fig. 3). These positive deflections were considerably smaller than the R or Rs waves recorded from uninfarcted ventricles during bundle branch block. Extensive transmural lesions of the ventricle with bundle branch block often yielded surface and intracavity complexes only one-third as large as those in corresponding leads from uninfarcted ventricles with bundle branch block.

(c) *Patchy Infarction.*—Before left bundle branch block was produced, epicardial leads recorded over patchy infarcts of the left ventricle exhibited QS complexes with embryonic R waves, QR deflections, or pure QS waves. The QS complexes usually differed from the cavity QS waves in magnitude, configuration, and often in timing. Both the epicardial and intracavity complexes became entirely or predominantly positive after the left bundle branch was blocked. The positivity of the epicardial surface was always greater than that of the cavity (Fig. 4). In ventricles with bundle branch block, the surface R wave recorded over patchy infarcts was smaller than the R wave obtained over normal muscle of the same heart and seemed larger than the R wave registered over through-and-through lesions.

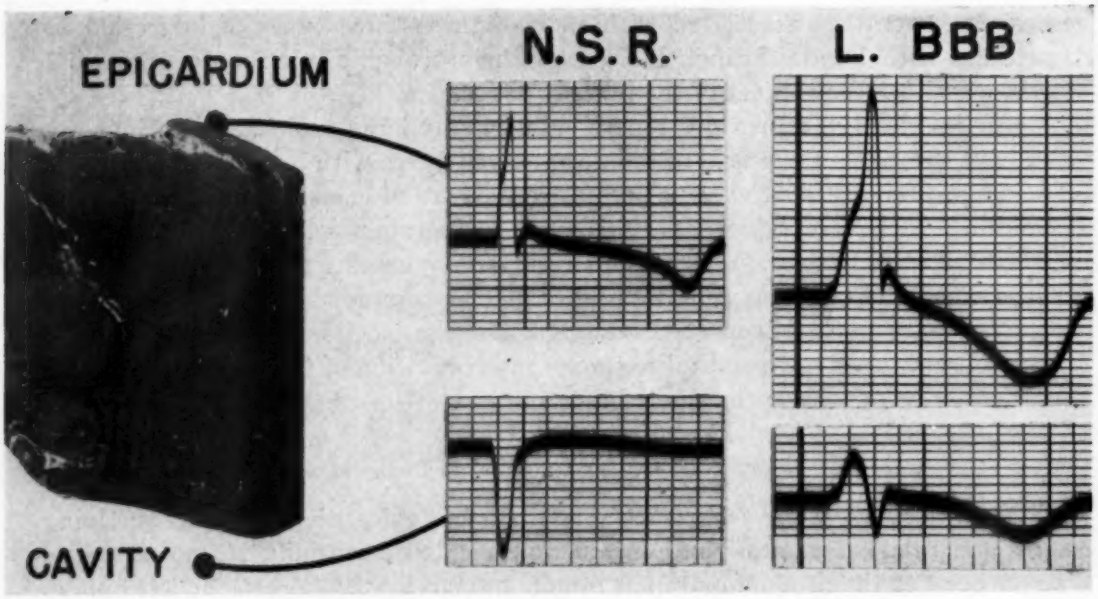

Fig. 1.—Surface and intracavity leads from uninfarcted portion of left ventricle recorded during normal intraventricular conduction (left) and after production of left bundle branch block (right). When block was produced, the normal epicardial R wave increased in amplitude and width, while the normal intracavity QS wave changed to a prolonged RS wave. Recorded on Sanborn Photographic Twin-Beam at paper speed of 75 mm. per sec. Each small box represents 0.013 sec.

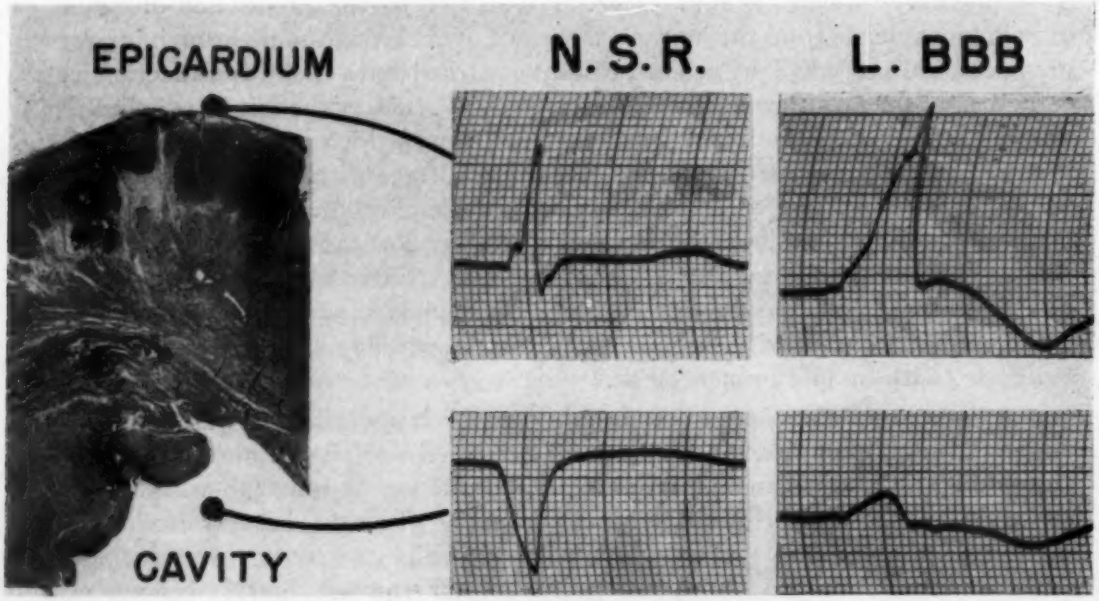

Fig. 2.—Surface and intracavity leads from portion of left ventricle containing subendocardial infarct. During normal intraventricular conduction, the intact epicardium over the lesion presented a normal Rs wave. The epicardial R wave became taller and wider when left bundle branch block was produced. Recorded on dual-channel Brush Recorder at paper speed of 125 mm. per sec. Each small box represents 0.008 sec.

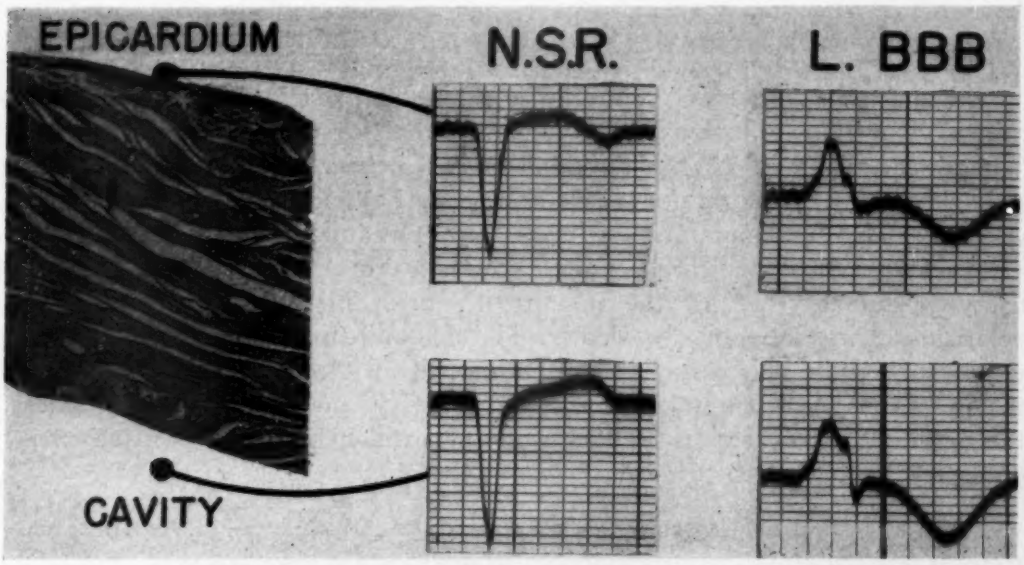

Fig. 3.—Record of same animal as in Fig. 1. Surface and cavity leads from region of through-and-through infarction in left ventricle recorded before and after left bundle branch block. Epicardial and intracavity complexes are almost identical, indicating that cavity potential is transmitted unaltered to epicardium. The coronary QS wave obtained during normal intraventricular conduction changed to a broad R wave after onset of left bundle branch block. Note that epicardial R wave registered during left bundle branch block is considerably smaller over transmural lesion than over normal portion of the same ventricle (compare this figure with Fig. 1).

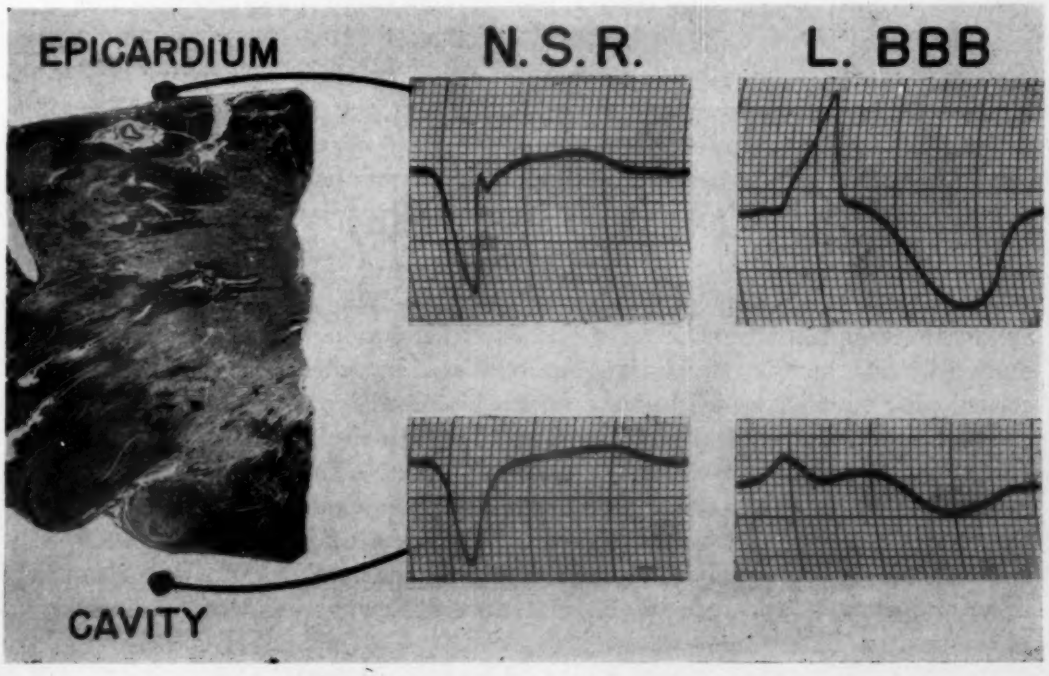

Fig. 4.—Epicardial and intracavity leads from region of patchy infarction in left ventricle. Before block, epicardial surface yields impure QS wave different from cavity QS wave. After block, epicardial complex consists of large R wave taller than the cavity complex. The R wave registered over region of patchy infarction in ventricle with bundle branch block is smaller than R waves recorded over regions of intact muscle (Fig. 1) or subendocardial infarction (Fig. 2) but is larger than R wave recorded over region of through-and-through muscle death (Fig. 3).

DISCUSSION

The preceding findings indicate that the strong positive potentials recorded on the epicardial surface of ventricles with bundle branch block are generated primarily by depolarization of the epicardial and subepicardial layers directly beneath the electrode. When the outer layers of the ventricle remained intact, as in subendocardial infarction, the surface potential during bundle branch block was the same as in uninfarcted ventricles. Patchy infarction which inactivated some, but not all, subepicardial muscle caused a diminution of the surface positivity, presumably because the amount of subepicardial tissue undergoing depolarization was abnormally small. Through-and-through infarction of the ventricle with bundle branch block eliminated all electrical activity between the epicardial electrode and the subjacent cavity; under such circumstances, the dead tissue serves as a conductor through which the small positive cavity potential is transmitted to the surface.

These conclusions concerning the predominant influence of the outer ventricular layers on the surface electrocardiogram are consistent with previously reported observations in normal hearts and in infarcted ventricles without bundle branch block. Recent experimental studies of the normal left ventricle have revealed that the deeper intramural layers are activated almost simultaneously.[4] Like the conducting system, the subendocardial region apparently transmits the depolarization process too rapidly to affect the electrocardiogram. Hence the normal surface complex must result from subepicardial depolarization. This observation was confirmed by removing or damaging the outer ventricular layers. Although the entire subendocardial region remained intact, the surface R wave became abnormally small or was replaced by a QS deflection.[5] Infarcts confined to the deeper layers of the myocardium, on the other hand, did not alter the normal surface complex.[5] In ventricles with normal conduction or with bundle branch block, therefore, the surface electrocardiogram appears to reflect primarily the status of subepicardial muscle and is relatively uninfluenced by deeper layers of the wall.

The present experimental study of epicardial potentials may have clinical applications in the diagnosis of myocardial infarction in the presence of left bundle branch block. Among the animals studied, the epicardial R wave recorded over ventricles with bundle branch block was considerably smaller when extensive transmural infarcts were present beneath the electrode than when the underlying subepicardial muscle remained intact. This would indicate that in patients with left bundle branch block, the development of a "hole" through the anterior wall of the ventricle should be manifested by an abrupt decline of the R wave in precordial leads from overlying sites. Patchy or less extensive anterior infarction theoretically should cause somewhat less drastic diminution of the positive potential.

We have observed several patients with left bundle branch block who developed marked reductions in the size of the left ventricular R wave in association with myocardial damage. In Fig. 5 are shown the tracings of a 57-year-old

male with arteriosclerotic heart disease. Following two earlier myocardial infarctions, the patient had a persistent left bundle branch block (Fig. 5,*A*). The tracings demonstrate a typical left bundle branch block with broad R waves over the left ventricle which are 7.5 mm. tall in V_5 and 7 mm. tall in V_6. The patient then suffered another myocardial infarction. The tracing taken after this attack is shown in Fig. 5,*B*. Note the marked reduction in the height of the R wave in the tracings from positions V_5 and V_6. The R wave in V_5 is now 3 mm., and in V_6 it is now 2 mm. In view of the experimental findings, these alterations in the R wave are best explained by the probable development of a through-and-through infarction, or more extensive patchy infarction with epicardial involvement.

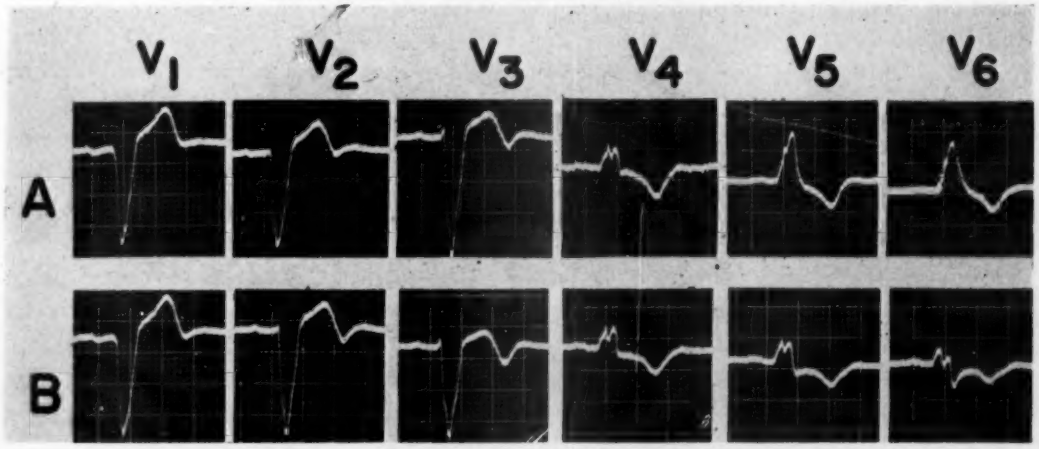

Fig. 5.—Precordial tracings from a patient with coronary artery disease and left bundle branch block. *A*, Six precordial leads taken shortly before the recent infarction. Note the height of the R wave in the V_5 and V_6 leads. *B*, Precordial tracings taken after recent infarction. Compare the height of the R wave in V_5 and V_6. Note the marked reduction in height of the R wave. Paper speed of 25 mm. per sec.

As described in the earlier paragraphs, the experiments demonstrate that the pathologic situations may be differentiated in direct surface leads by comparing the magnitude of local depolarization potentials recorded over different parts of the lesion. Unfortunately, the electrocardiographic diagnosis of myocardial infarction in patients with left bundle branch block is considerably complicated by limitations of the precordial leads as well as by the nature of clinical infarcts. It had been noted by earlier workers[2] that there will be instances of decreases in the surface potential due to the development of infarcts in the ventricular wall which might be perceptible in direct leads but may not be perceptible in precordial tracings. This is particularly true of infarcts with a small amount of subepicardial involvement.

Nevertheless, it is noteworthy that the value of precordial leads in detecting myocardial infarction in the presence of left bundle branch block will be greatly enhanced when multiple precordial leads are taken or if a control electrocardi-

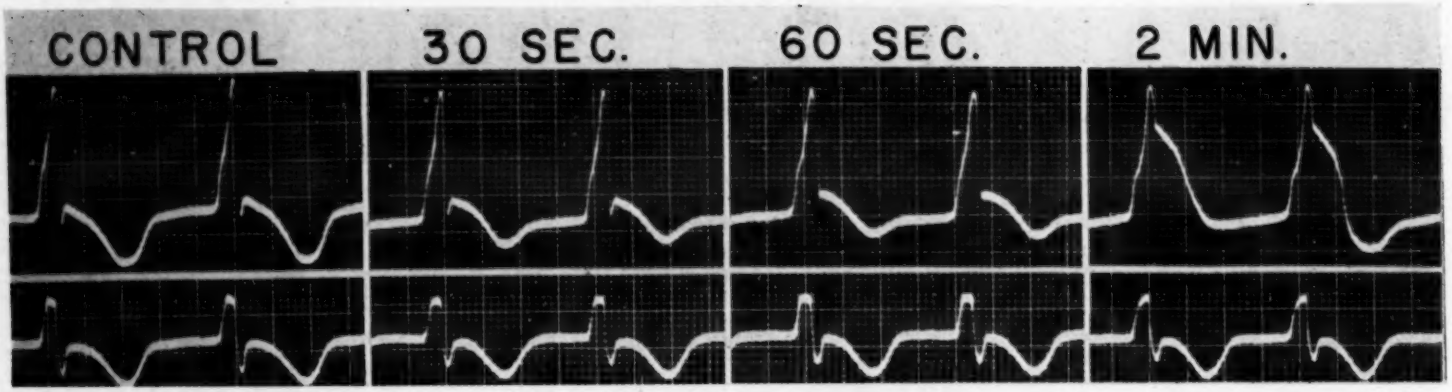

Fig. 6.—Simultaneous tracings recorded before and after a coronary artery tie from the epicardium (upper tracings) and subendocardium (lower tracings) of dog's left ventricle with left bundle branch block. The control tracings both show an isoelectric S-T segment. Following coronary artery tie there is elevation of the S-T segment in the surface leads. The S-T segment in the leads from the subendocardium remain isoelectric at all times. Tracings recorded on Sanborn Poly-Viso at paper speed of 50 mm. per sec.

ogram taken before infarction is available. If one precordial lead shows abnormally small R waves while the adjacent precordial leads show larger positive complexes diagnostic of left bundle branch block, an infarct should be suspected beneath the region from which the small deflections are obtained. If the electrocardiograms following suspected myocardial damage show a marked reduction in the height of the R wave as compared with preinfarction control tracings, then the diagnosis of infarction is confirmed. If such lesions involve the anterior wall of the blocked ventricle and an abnormally small R wave should occur in the left precordial leads, posterior infarcts involving a considerable area of subepicardial muscle should decrease the R wave in aV_F.

B. S-T SEGMENT

The preceding observations modify previously known clinical evidence that the changes of the QRS complex in the precordial leads may be absent or of equivocal meaning when myocardial infarction complicates left bundle branch block. Nevertheless, in the early diagnosis of acute myocardial infarction, the significant changes are usually limited to the RS-T segment. This section will concern several observations on changes of the S-T segment in the presence of left bundle branch block.

Attention must first be directed to those S-T and T-wave changes that exist secondary to the left bundle branch block and are not dependent upon coronary artery disease. In uncomplicated left bundle branch block, the S-T segment and T wave usually move in a direction opposite to the QRS complex. In those leads where there is a tall, broad R deflection, the onset of the S-T segment is usually depressed and the T wave inverted. In those leads where there is a prominent wide S wave, there is usually upward displacement of the R-ST junction together with an upright T wave. Dressler and associates[7] found that in Positions C_2 and C_3 this elevation may be as high as 7 mm. and the upright T wave might have an amplitude of 19 mm.

Experimental Observations.—In order to understand more fully the S-T segment changes in the presence of left bundle branch block, a series of experiments were performed in which simultaneous epicardial and subendocardial electrocardiograms were recorded when acute myocardial infarction was superimposed on left bundle branch block.

Left bundle branch block was produced in five animals in a manner previously described. Control electrocardiographic tracings were taken from the epicardial, subendocardial, cavity, and limb leads. All intramural tracings were recorded only after the S-T segment changes resulting from placement of the electrodes had disappeared. After the control tracings, one of the large branches of the anterior descending coronary artery was tied. This resulted in an immediate area of cyanosis of variable portions of the left ventricle and included the region in which the electrodes were placed. Tracings were taken at 30-second intervals following the occlusion of the coronary artery.

The results of a typical experiment are shown in Fig. 6. The control tracings demonstrate left bundle branch block in simultaneous tracings recorded from the epicardium and underlying subendocardial areas of the left ventricle. The

remaining tracings were recorded at 30-second intervals after a branch of the left descending coronary artery which supplied the area of myocardium from which the tracings were being recorded was tied. In the electrocardiograms taken 30 seconds after the tie, the surface lead shows S-T segment elevation while in the simultaneous subendocardial lead the S-T segment is isoelectric. One minute after the tie, the S-T segment elevation in the surface lead is still more pronounced while there is still no change in the subendocardial lead. At two minutes, the elevation in the epicardial lead is very marked with no change in the subendocardial lead.

DISCUSSION

Despite the presence of left bundle branch block, the experiments demonstrate some of the S-T segment alterations that occur with acute myocardial damage. It was found that the S-T segment changes of acute damage are more pronounced in the outer layers of the myocardium than in the inner layers. This was in keeping with previous observations on the S-T segment.[4]

With acute ischemia there develops in the direct left ventricular leads S-T segment elevation replacing any depression that might be present secondary to the bundle branch block. As shown previously by Sodi-Pallares and associates,[8] this positive S-T displacement may appear in the left precordial leads and allow the diagnosis of a recent infarction superimposed on a left bundle branch block. Positive displacement of the S-T segment in aV_F suggested the diagnosis of infarction of the diaphragmatic aspect of the heart. Thus the changes which occur with the acute injury, i.e., S-T elevation, are primarily the same when bundle branch block is present as when normal conduction is present.

The S-T segment changes in the electrocardiogram which occur with exercise are considered to be an important diagnostic aid in the diagnosis of coronary artery disease. There has been in the past a great deal of information gathered concerning what constitutes significant changes in the presence of normal conduction. However, there have been relatively few observations made on the electrocardiographic changes which occur with exercise in the presence of left bundle branch block. Such observations should be of value not only in diagnosing coronary artery disease in the presence of left bundle branch block but might aid in the better understanding of other S-T segment changes.

Recently we performed a two-step exercise test on a patient with bundle branch block. He had been followed over a 10-year period with twice yearly checkups. Throughout the period, repeated electrocardiograms demonstrated left bundle branch block. There had been no symptoms of heart disease until recently when he developed angina pectoris. Before exercise, the control electrocardiogram revealed the left bundle branch block with S-T depression of 1 mm. in Lead I; S-T elevation of 4 mm. with upright T wave in V_2; and S-T depression of 0.5 mm. and a biphasic T wave in V_6. Immediately following a two-step exercise test, the depression in Lead I became 2 mm.; V_2 showed S-T elevation of 7 mm. with a taller, more peaked T wave; and V_6 showed 1.5 mm. depression.

The changes noted in this case are similar to those noted by Feil and Brofman[9] when exercise was performed on patients with bundle branch block and

angina pectoris. Thus it appears that S-T segment changes may at times be utilized to substantiate a diagnosis of coronary artery disease even in the presence of left bundle branch block. The changes in the S-T segment are similar to those which occur in the absence of left bundle branch block. Depression occurred in Lead I and in the precordial leads over the left ventricle following exercise despite the presence of left bundle branch block.

Several patients under our observation have spontaneously developed S-T segment changes similar to those which occurred on the exercise test. These alterations in the S-T segment occurred with attacks of chest pain more severe than the ordinary attack of angina pectoris. There were no signs of acute myocardial infarction, and it was believed that these represented episodes of coronary insufficiency or coronary failure. Psychogenic or other causes of alteration of the S-T segment must always be eliminated by clinical judgment.

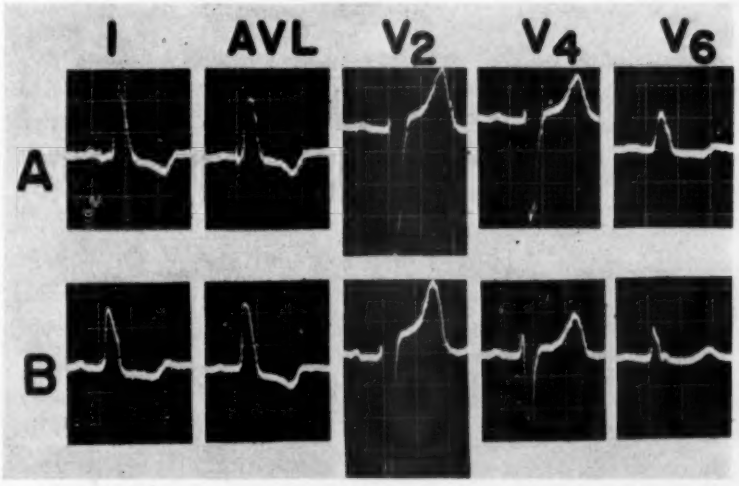

Fig. 7.—Tracings from a 65-year-old woman with arteriosclerotic heart disease and left bundle branch block. *A*, Control, base line tracings with left bundle branch block. *B*, Tracings following an episode of coronary insufficiency. In the latter tracings, note the S-T segment depression in Leads I and aV_L together with the more marked S-T segment elevation in V_2. Tracings recorded at paper speed of 25 mm. per sec.

A 65-year-old lady was under observation for 12 years because of arteriosclerotic heart disease with left bundle branch block. An electrocardiogram shown in Fig. 7 demonstrates the left bundle branch block which she showed for many years. On Oct. 28, 1954 the patient was seen because of chest pain of several hours' duration. The pain was similar to other attacks of chest pain which she had suffered but was of longer duration. The electrocardiogram disclosed that in comparison to the previous tracings there was now S-T segment depression in Lead aV_L and S-T segment elevation in the leads from the right side of the heart.

This depression of the S-T segment in the leads overlying the left ventricle in combination with S-T segment elevation in leads overlying the right ventricle

has been observed in several other patients with left bundle branch block who had similar episodes of chest pain. In some patients, the change was most apparent in the right ventricular leads and did not appear or was very slight in the routine leads reflecting left ventricular activity. This is, of course, due to the fact that the ischemic area may be so located as to cause little or no perceptible changes in such leads as aV_F or aV_L. In many instances, however, the ischemic area may be well reflected in the left precordial leads, and the depression will appear reciprocal to the elevation in the right precordial leads.

In several cases, as in the preceding case, the S-T segment changes following the episode of coronary insufficiency are still within the limits of what are usually accepted as the limits of normal secondary RS-T segment changes of left bundle branch block. This again illustrates the great value of having available control and serial electrocardiograms so that these deviations in the S-T segment can be readily detected.

SUMMARY

Despite the absence of the coronary Q wave, there appear to be instances when myocardial infarction can be diagnosed in the presence of left bundle branch block. The experimental evidence showed that with a transmural infarction or patchy infarction with extensive surface damage there occurs a marked reduction in the size of the R wave recorded in the direct lead from that region. In some instances, the reduction in the R wave may be reflected in the corresponding precordial lead. Changes in the magnitude of the R wave in the left precordial leads in left bundle branch block will frequently be detected only if control tracings are available for comparison or if multiple precordial leads are taken.

Acute myocardial injury results in elevation of the S-T segment despite the presence of left bundle branch block. Thus, the diagnosis of acute myocardial infarction can be made from the finding of significant S-T segment elevation following the large, broad, positive complex of left bundle branch block. Other changes in the coronary circulation may also be reflected by alterations in the RS-T segment despite the presence of left bundle branch block. In our experience, coronary insufficiency as well as a positive two-step exercise test has resulted in changes in the S-T segment.

It would appear that the popular opinion that coronary artery disease cannot be diagnosed in the presence of left bundle branch block should be modified. It is true that there is absence of the coronary Q wave in myocardial infarction, but the previously discussed changes should aid in making more conclusive the diagnosis of coronary artery disease in the presence of left bundle branch block.

CONCLUSIONS

1. Bundle branch block was produced in nine dogs by cutting the left branch of the bundle of His after the experimental production of healed left ventricular infarcts.

2. Histologic examination of the infarcts revealed three different pathologic patterns: (a) lesions involving the subendocardial region only;

(b) through-and-through infarction consisting of uniformly dead tissue extending from endocardium to epicardium; and (c) patchy infarction involving all intramural levels but containing variable amounts of viable muscle.

3. Prior to the production of left bundle branch block, purely subendocardial lesions presented normal R or Rs waves in surface leads; through-and-through infarcts yielded surface QS waves identical with the cavity complex: patchy infarcts exhibited surface QS complexes with embryonic R waves, QR deflections, or pure QS waves differing from the cavity QS in magnitude, configuration, and often in timing.

4. After bundle branch block was produced, leads from the surface of all three types of lesions displayed pure R or Rs waves. Subendocardial infarcts yielded abnormally tall, broad complexes indistinguishable from those recorded over the uninfarcted myocardium. Through-and-through infarcts presented small R or Rs waves identical with the intracavity deflection. Patchy infarcts exhibited positive epicardial complexes smaller than those found over uninfarcted ventricles with bundle branch block but larger than those obtained over through-and-through lesions of ventricles with bundle branch block.

5. The positive potential registered at the epicardial surface of ventricles with complete bundle branch block is derived primarily from depolarization of the outer layers of the ventricle and is not detectably affected by deeper layers of the myocardium.

6. In patients with bundle branch block, the development of myocardial infarcts involving large amounts of subepicardial muscle may be manifest by a decrease in the magnitude of the R wave in precordial leads overlying the left ventricle or aV_F. The value of preinfarction tracings for comparison is discussed.

7. Experimental studies in five dogs disclosed that the S-T segment changes which occur in direct leads in the presence of left bundle branch block are produced primarily by the outer layers of the ventricular myocardium.

8. Acute myocardial infarction in the presence of left bundle branch block will frequently be manifest in patients by S-T elevation in the precordial leads overlying the left ventricle or aV_F.

9. In the presence of coronary artery disease, S-T segment changes may occur despite the presence of left bundle branch block. With exercise or with spontaneous coronary failure, S-T segment depression occurs in those leads overlying the left ventricle or aV_F, and RS-T elevation occurs in those leads overlying the right ventricle. The S-T elevation in V_1 through V_3 may be the more obvious change in such circumstances.

10. The S-T segment changes may be detected only if the previous tracings are available for comparison.

SUMMARIO IN INTERLINGUA

Esseva facite in animales observationes in re chronic infarcimento myocardiac complicate per bloco del branca sinistre.. Le unda epicardial R, registrate supra ventriculos con bloco de branca esseva considerabilemente plus parve quando extense infarctos transmural esseva presente infra le electrodo que

quando le subjacente musculo subepicardial remaneva intacte. Correspondentemente, in patientes con bloco de branca sinistre, le disveloppamento de un infarcimento pote manifestar se per un abrupte descendita del unda R in derivationes precordial ab sitos suprajacente. Es presentate casos clinic pro illustrar le valor diagnostic de iste puncto.

Acute vulneration myocardial in animales resultava in le elevation del segmento S-T in despecto del presentia de bloco del branca sinistre. Similemente, in patientes human, acute infarcimento myocardial in le presentia de bloco del branca sinistre se manifesta frequentemente per elevation del segmento S-T in le derivationes precordial suprajacente. Insufficientia coronari e etiam un bi-phasic test a exercitio pote resultar in significative alterationes del segmento S-T in despecto del presentia de bloco del branca sinistre. Iste alterationes pote esser detegite solmente si previe registrationes es disponibile como base del comparation.

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TALL PRECORDIAL T WAVES AS THE EARLIEST SIGN IN DIAPHRAGMATIC WALL INFARCTION

FRED W. WACHTEL, M.D., AND EUGENE M. TEICH, M.D.

NEW YORK, N. Y.

THE importance of early recognition of an acute myocardial infarction is self-evident. With the advent of routine electrocardiography, it has been possible in most instances to confirm the clinical impression of acute coronary occlusion. The earliest changes in this condition have been observed in the QRS and RS-T segments of the electrocardiogram. More recently, attention has been drawn to T wave changes as the initial abnormality in myocardial necrosis.

Pardee,[1] in one of the earliest descriptions of the electrocardiographic pattern in posterior wall infarction (more correctly referred to as diaphragmatic or inferior wall infarction), stated that the T wave is usually of larger size than customary and shows a somewhat sharper peak. It should be noted that concurrent RS-T elevations were present in the published electrocardiograms. Examples of tall T waves commonly found in standard Leads II and III of the "Q_1-T_1 type" were observed by Bohning and Katz.[2] They were referred to as "upright coronary T waves" and were thought to be the mirror image of the coveplaned T wave seen in Lead I. Cases were observed within twelve hours after the onset of pain by Dressler and Roesler,[3] in which high T waves were the outstanding feature in the earliest stage of anterior myocardial infarction, when elevations of the RS-T segment and QRS changes were absent. Later these high T waves became isoelectric, diphasic, or inverted as the pattern of anterior wall infarction evolved.

Barker[4] describes a case in which the earliest electrocardiographic evidences of myocardial infarction were tall, sharply pointed T waves in precordial Leads V_{2-6}. These T waves later became semi-inverted, finally coveplaned and deeply inverted at a time when Q waves had appeared in standard Leads I and II, aV_L, aV_F, and precordial Leads V_5 and V_6. An rS pattern was also present in precordial Leads V_{1-4}. These changes are indicative of anterolateral infarction, with some diaphragmatic involvement.

In animal experiments performed by Bayley and associates,[5] in which they occluded the anterior descending branch of the left coronary artery in dogs, elevated RS-T segments and tall peaked T waves constituted the first stage in the electrocardiographic evolution of anterior wall infarctions. These findings were later confirmed by Graham and Laforet[6] in humans. During mitral val-

From the Department of Medicine, The Mount Sinai Hospital, New York, N. Y.
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vuloplasty, these observers noted an increase in the height of the T waves occurring five minutes after accidental ligation of the left main coronary artery in two patients. Although no precordial leads were available for inspection, the tracings were compatible with an anterior wall infarction.

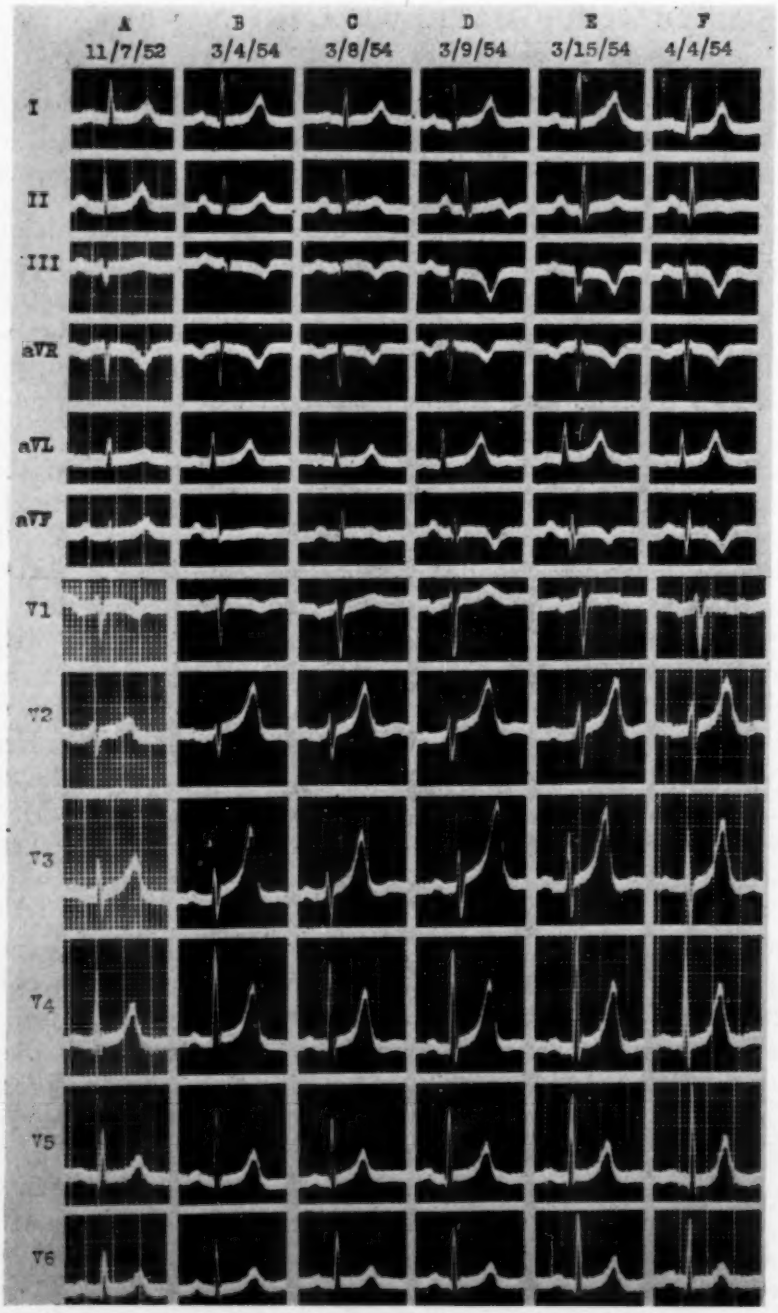

Fig. 1.—Case 1. *A*, Normal ECG 16 months prior to acute episode. *B*, Seven days after onset of precordial pain, showing remarkably tall peaked T waves in V_{2-5}. No diagnostic changes in other leads. *C*, RS-T elevations in Leads II, III and aV_F; T_3 inverted, TaV_F diphasic. *D*, Q waves and diphasic T_3 and TaV_F. *E and F*, Q_2, Q_3, QaV_F with inverted T_3 and TaV_F. Tall T in V_{2-4}.

Hellerstein and Leibow,[7] and Wolff[8] amongst others, have observed high peaked precordial T waves in acute diaphragmatic wall infarction. These have been observed at the same time that RS-T elevations and T wave inversions appeared in standard Leads II and III, and aV_F, as well as reciprocal RS-T depressions in Leads I, aV_L, and right-sided precordial leads. In none of these cases did precordial tall T waves precede the presence of Q waves or RS-T elevations in Leads II, III, and aV_F.

In posterolateral and true posterior wall infarction, tall R waves and tall pointed T waves are often present in V_{1-2}.[9-11]

High peaked T waves in the precordial leads have been observed in acute myocardial infarctions of the following types:

1. In the very early stages of anterior wall myocardial infarction.
2. In diaphragmatic infarction, accompanying significant Q waves and/or RS-T segment elevation and T wave inversion in Leads II, III and aV_F.
3. In true posterior infarction at the same time that tall R waves were present in the same leads.

We have observed two cases in which the earliest significant changes in the electrocardiogram were tall peaked T waves in precordial Leads V_{2-5} at a time when the clinical picture was compatible with myocardial infarction. Serial electrocardiograms in these cases revealed the evolutionary pattern of acute diaphragmatic infarction.

CASE 1.—This was the third Mount Sinai Hospital admission of S.K., a 60-year-old white man, who entered on March 4, 1954, with complaints of intermittent chest pain of ten days' duration.

The patient was well until February, 1952, when he noted the onset of left chest pain, with radiation to the left arm. It was atypical of angina in that it was not induced by effort, but the pain could be evoked by pressure upon the left supraclavicular fossa. X-ray examination of the left shoulder at that time demonstrated the presence of a large calcific density in the vicinity of the insertion of the supraspinatus tendon. There was also degenerative arthritis of the cervicodorsal spine. An electrocardiogram taken at this time was interpreted as normal (Fig. 1,*A*). In the intervening period, from 1952 to this admission, the patient continued to have occasional fleeting episodes of chest pain. The remainder of the history was noncontributory.

Three days prior to admission, the chest pain became very severe, crushing in nature, located in the left hemithorax, with radiation down the left arm. The pain was not relieved by nitroglycerin, but responded to a narcotic. Recurrence of the pain prompted hospitalization of the patient.

The physical examination was within normal limits, except for blood pressure of 170/90 mm. Hg. An electrocardiogram was taken the day of admission (Fig. 1,*B*). During the next four days, he experienced several episodes of chest pain, requiring a narcotic for relief. At no time was there any change in the pulse rate, blood pressure, or physical examination.

The initial hemoglobin was 14 Gm., white blood count 10,100, with a normal differential. The erythrocyte sedimentation rate was 35 mm./hour Westergren. During the entire hospital stay, the ESR ranged between 32 and 38 mm./hour. The BUN was 10 mg. per cent and the total cholesterol 310 mg. per cent.

The hospital course was uneventful and the patient was discharged after six weeks. A one-year follow-up found the patient asymptomatic.

CASE 2.—L.C., a 57-year-old merchant, was admitted to The Mount Sinai Hospital for the first time on March 13, 1954, complaining of severe, radiating substernal chest pain of five days' duration.

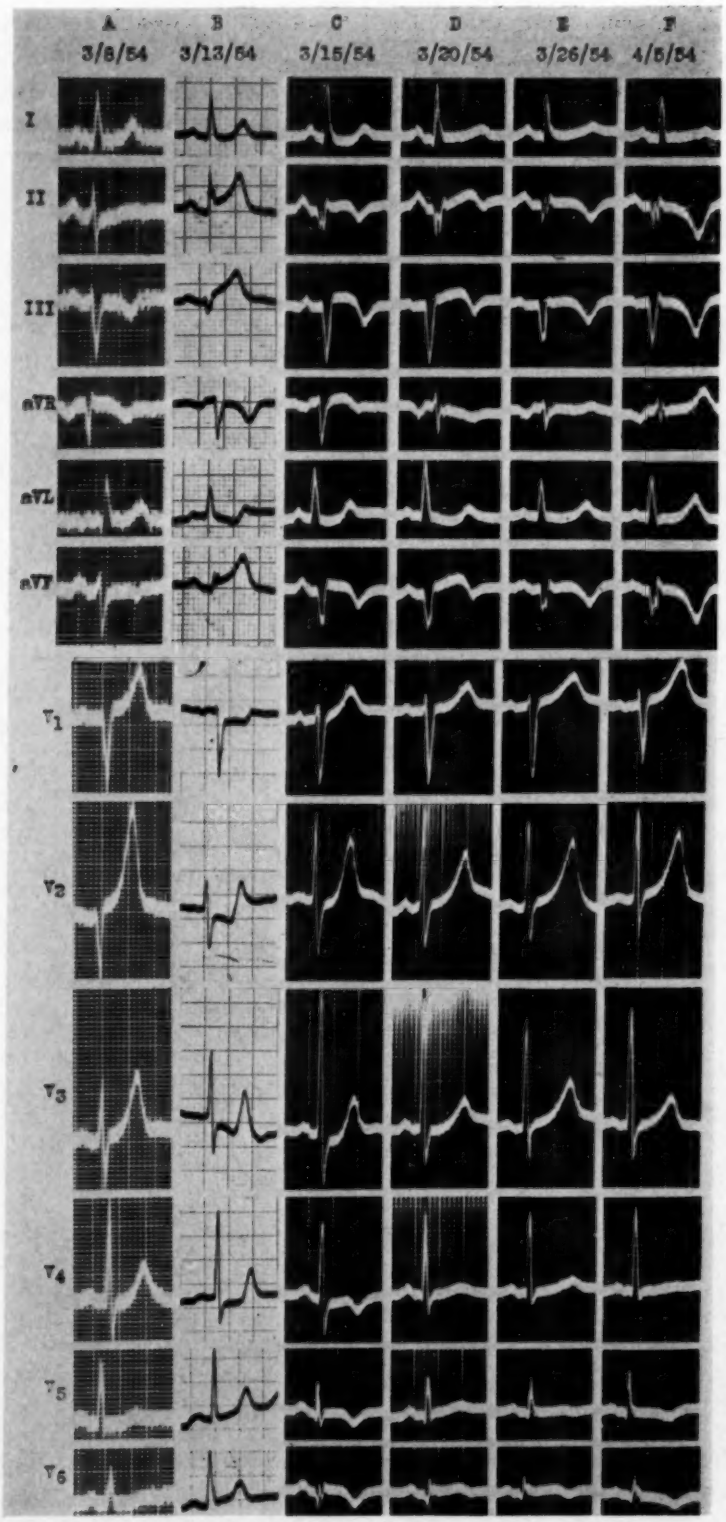

Fig. 2.—Case 2. *A*, Shortly after onset of chest pain revealing huge peaked T waves in V_{2-4}. T_3 and TaV_F are slightly inverted, but not diagnostic. *B*, Marked RS-T elevations in Leads II, III and aV_F with reciprocal RS-T depressions in aV_R, V_{1-4}. *C*, Deep Q_2, Q_3, QaV_F, QV_6, elevated RS-T segments and coveplaned, deeply inverted T waves in Leads II, III, aV_F and V_6. *D, E, and F*, Deep Q_2, Q_3, QaV_F, small Q_{V6}, and deeply inverted T_2, T_3, TaV_F, and T_{V5-6}. Note tall R wave in V_{2-3} indicative of true posterior involvement.

The patient had intermittent, transient substernal oppressive sensations with radiation to the neck, aggravated by exertion and relieved by rest, for at least two years. Five days prior to admission, he had severe substernal pain with radiation to the back, neck, left arm, and forearm. He was seen by a physician who administered a narcotic for pain after an electrocardiogram was taken (Fig. 2,*A*). His pain subsided after several hours but recurred the next day as a dull precordial ache, which persisted until the time of admission.

On the night of admission, a physician was called because of the unrelenting anterior chest pain, constricting in nature with characteristic radiation. An electrocardiogram was taken just prior to admission (Fig. 2,*B*).

There was no previous history of coronary occlusion, diabetes, or hypertension. Systemic review was unremarkable.

Physical examination revealed a well-developed, somewhat obese man, quite apprehensive, diaphoretic, and complaining of substernal chest pain. Temperature on admission was 98.0°F., but rose to 101.2°F. on the next day. Pulse was 88 and regular; blood pressure, 116/80 mm. Hg in both arms. The heart was not enlarged. The tones were distant. Rhythm was regular and no murmurs or rub were heard. The remainder of the physical examination was within normal limits.

Laboratory examination at the time of admission revealed the hemoglobin to be 15.1 Gm., and W.B.C. 10,900, with a moderate shift to the left. The erythrocyte sedimentation rate was 49 mm./hour Westergren and rose to 79 mm./hour after one week. The ESR remained elevated to 63 mm./hour at the time of discharge. Leukocyte count returned to normal after two weeks. Repeated urinalyses were normal. Total serum cholesterol was 200 mg. per cent. Chest x-ray just prior to discharge revealed a tortuous, elongated aorta, but no other abnormalities of the heart or lungs.

The course in the hospital was uncomplicated. A temperature elevation between 100°-101°F. was recorded for the first six days of hospitalization but was normal for the remainder of the hospital stay. Shortly following admission, the patient complained of moderate substernal discomfort and later of left shoulder pain which required narcotics for relief. His pain subsided after the first week and did not recur. After slow progressive ambulation, he was discharged in the seventh hospital week.

The patient is now one year post-acute infarction and has continued to do well.

DISCUSSION

Analysis of the electrocardiograms taken at the onset of symptoms (Figs. 1,*B*, 2,*A*) reveals changes only in the T waves of Leads V_{2-5}. These T waves are unusually tall and peaked. The QRS and T wave complexes in the other leads are not unusual. In the next tracings (Figs. 1,*C*; 2,*B*) there is diagnostic evidence of diaphragmatic infarction, as shown by RS-T elevations and T wave changes in Leads II, III, and aV_F; namely, inverted T waves in Case 1 and upright T waves in Case 2. The remainder of the tracings reveals the evolutionary pattern of typical diaphragmatic occlusion in both cases, with some lateral and posterior involvement in Case 2. In the later tracings, the tall pointed T waves in V_2 and V_3 are present concurrently, with deeply inverted and coveplaned T waves in Leads III and aV_F in Case 1 and Leads II, III, and aV_F in Case 2.

In retrospect, these patients when first seen following the onset of chest pain were in the "premonitory" or "impending" stage of myocardial infarction.[12,13] Although the classical electrocardiographic changes encountered in myocardial ischemia are RS-T depressions and/or T wave inversions, we believe that the tall peaked T waves observed at the onset of the coronary episodes in our patients are indicative of ischemia of the diaphragmatic and posterior portions of the

left ventricle. From analysis of the first electrocardiograms, it can be seen that the predominant T wave forces are directed principally anteriorly and slightly superiorly. Thus, leads showing maximal RS-T segment depression and T wave inversion could be observed only in posteriorly and inferiorly located leads. There are no leads corresponding to these positions in the conventional 12-lead electrocardiogram. However, precordial leads V_{2-5} are at the mirror image position to the theoretical ones previously described. Therefore, the tall peaked T waves in V_{2-5} are reciprocal reflections of T wave inversions and indicate ischemia of the diaphragmatic and posterior regions of the heart. Only later, with the appearance of deep Q waves, did the T wave forces assume a more superior orientation. This appears as deeply inverted T waves in Leads II, III, and aV_F which are the commonly observed changes in diaphragmatic wall infarction.

Tall, sharply pointed T waves have been observed in a variety of normal and pathologic states. When these occur in an adult following chest pain, acute myocardial damage should be suspected. Further observation and follow-up electrocardiograms are mandatory.

SUMMARY

Two cases are presented in which the earliest electrocardiographic evidence of myocardial necrosis was tall, sharply pointed T waves in precordial Leads V_{2-5}. The mechanism of their production is discussed. Serial electrocardiograms revealed the evolutionary pattern of diaphragmatic wall infarction.

SUMMARIO IN INTERLINGUA

Alte, acute undas T precordial ha previemente essite observate in acute infarcimento myocardial diaphragmatic, accompaniante significative undas Q e/o elevate segmentos RS-T e inversion del unda T in le derivationes 2, 3, e aV_F. Es describite duo casos in que le prime indicios electrocardiographic de necrosis myocardial esseva alte, acute undas T in le derivationes precordial V_{2-5}, sin ulle altere alterationes significative. Iste constatationes indica in nostre opinion le presentia de ischemia del superficies diaphragmatic e posterior del ventriculo sinistre. Electrocardiogrammas serial revelava le configuration classic de infarcimento del pariete diaphragmatic.

Quando alte, acute undas T precordial occurre in adultos in association con dolores thoracic, acute lesion myocardial debe esser prendite in consideration.

We are indebted to Dr. Morton H. Edelman and Dr. Morris F. Steinberg for permission to use the clinical material presented. Sincere thanks are due Dr. Arthur Grishman for his suggestions in the preparation of this paper.

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PULMONARY RESECTIONAL SURGERY IN MITRAL STENOSIS

RICHARD WALLYN, M.D., ROBERT DILLON, B.S., M.D.,* AND
BERNARD L. BROFMAN, M.D.

CHICAGO, ILL.

A CASE of mitral stenosis and misdiagnosed oat-cell carcinoma is presented. The occlusion of the right pulmonary artery by a balloon during cardiac catheterization was used to show pulmonary resectional tolerance in this situation. This was also shown to be another method of determining pulmonary "capillary" pressure. Persistence of pulmonary vasomotor lability is demonstrated even in the presence of pulmonary hypertension due to mitral stenosis. Unduly prolonged obstruction with angiography may produce significant pathology in the obstructed lung. We are presenting this case to: (1) document with physiologic data the hemodynamics of simulated pneumonectomy in mitral stenosis and (2) illustrate a preoperative technique for determining pulmonary resectional tolerance.

METHOD

Catheterization was done by the technique of Cournand and associates,[1] with a special catheter to be described. Pulmonary wedge pressure was determined by the method of Hellems and associates.[2] Pressures were recorded on a multichannel direct-writing oscillograph† through an electromanometer. Mean pressures were obtained by electric integration. Blood oxygen contents were determined on the Van Slyke apparatus. Oxygen consumption was determined by the open circuit method, using a Pauling oximeter and Haldane apparatus. Cardiac output was determined by the direct Fick principle. Pulmonary functions were done by the method of Warring.[3] Pulmonary artery occlusion was accomplished by the balloon catheter method of Carlens and associates,[4] elaborated on by Hanson.[5] A triple-lumen catheter of approximately 9F size, with a latex balloon on the middle lumen (as designed by one of us, B.L.B.), was used,[6] obviating the need for a second catheter.

From the Hektoen Institute of Medical Research, the Cardiovascular Laboratory of Cook County Hospital, the Department of Medicine of the Stritch School of Medicine of Loyola University, and the Cardiovascular Laboratory of Mt. Sinai Hospital, Cleveland, Ohio.

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*Post Doctorate Fellow in Cardiology, United States Public Health Service.

†Sanborn Company, Cambridge, Massachusetts.

CASE REPORT

A 40-year-old white male factory worker was first seen at Cook County Hospital on March 16, 1954. There was a history of gradual onset for two years of limitation of activity, and a two-month history of dyspnea on exertion, orthopnea, nocturnal dyspnea, and productive cough. For a few days preceding admission there had been blood streaking of the sputum. Past history revealed no symptoms of rheumatic fever. In 1942 he had been rejected for Army service because of a heart murmur. Physical examination revealed a brachial blood pressure of 120/80 mm. Hg, a regular pulse of 80 per minute, and a temperature of 100.4° F. orally. The neck veins were not distended. The heart was not enlarged to percussion. The second heart sound was louder over the pulmonary area. There was an aortic Grade 2 systolic murmur and an apical Grade 3 systolic

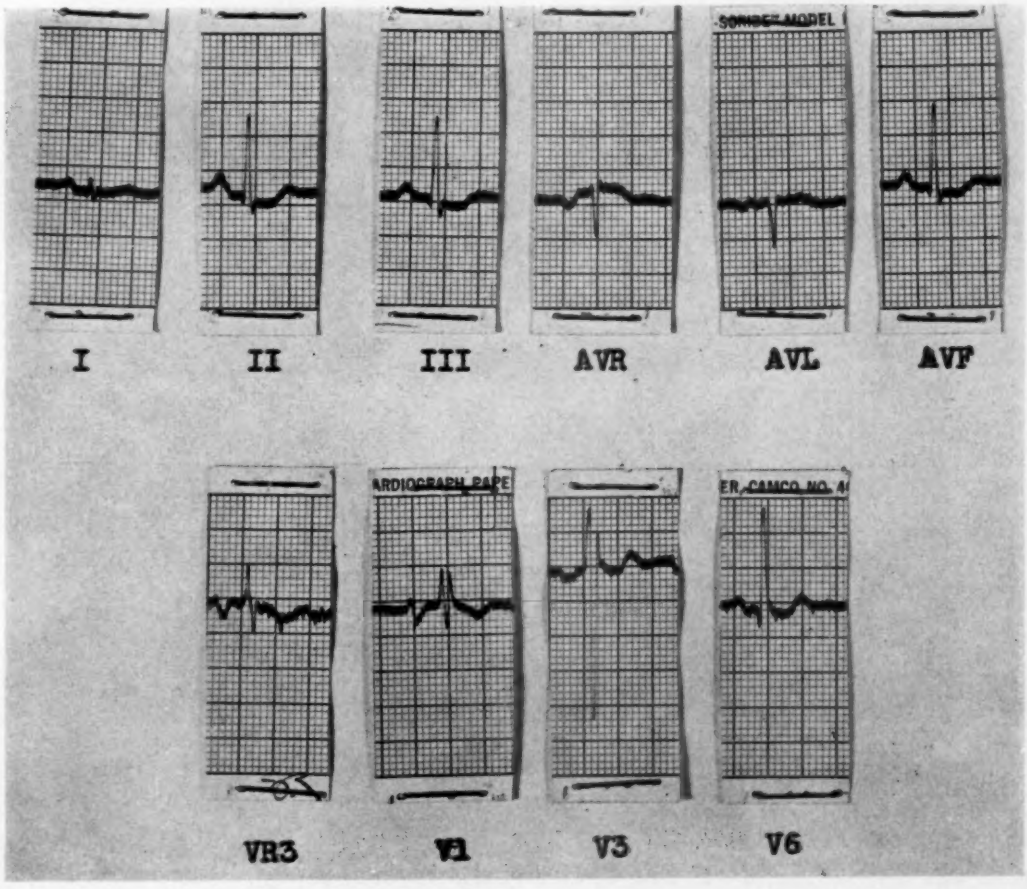


Fig. 1.—Electrocardiogram. .

plus a diastolic murmur. Auscultation of the lungs revealed basal râles. The liver was palpable two finger-breadths below the costal margin. Urine, serology, and N.P.N. were normal. Blood culture revealed only diphtheroid contaminants. X-ray examination was interpreted as revealing an inflammatory process in the right lung base, involving the right middle and lower lobes. There was fluid in the left base. Pulmonary vascular markings were increased. The heart was of normal size, with a prominent pulmonary artery segment. The impression was rheumatic heart disease in failure. Digitalization resulted in a good response. The course was afebrile and the patient was discharged on digitalis maintenance after twelve days of hospitalization.

The patient was readmitted on April 23, 1954, for hemoptysis of bright red blood. This recurred in amounts up to 150 c.c. while he was in the hospital. Physical examination at that time revealed a blood pressure of 100/60 mm. Hg, a regular pulse of 84 per minute, respiration at 18 per minute, and a temperature of 99.0° F. orally. No petechiae were seen in the skin or mucosal surfaces. There was no neck vein distention. The liver was palpable two finger-breadths below the right costal margin. Examination of the chest revealed dullness over the left base and wheezes over the right side anteriorly. The cardiac examination revealed a mid-diastolic murmur at the apex with a loud mitral first sound. There was also an aortic systolic murmur which radiated well into the neck. The stool benzidine reaction was negative. Urine, serology, C.B.C.,

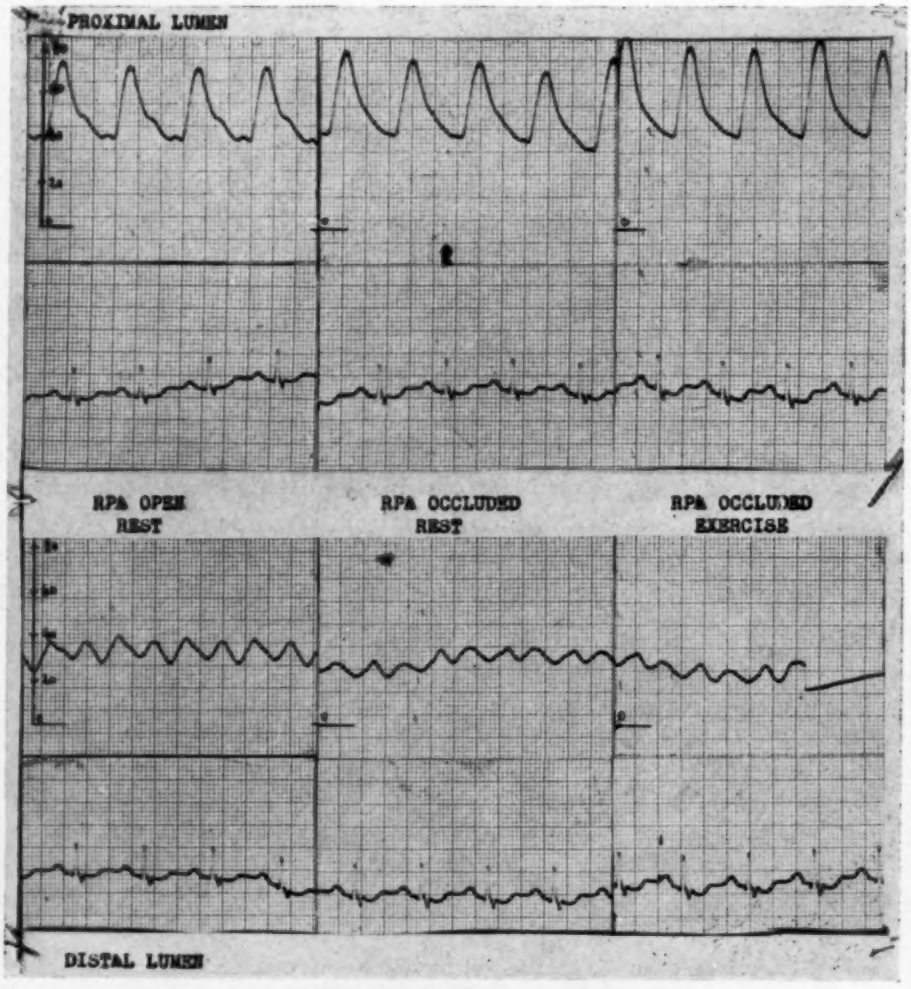


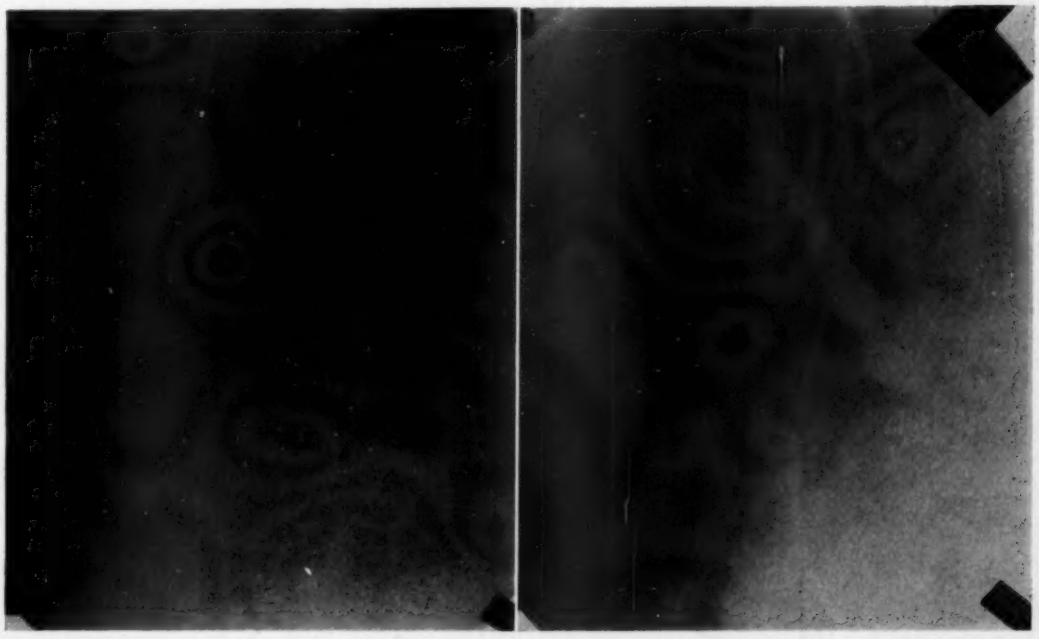
Fig. 2.—Pressure tracings from pulmonary artery.

N.P.N., and total protein were normal. An electrocardiogram (Fig. 1) revealed an RSR′ pattern in V_1. Venous pressure was 120 mm. of saline at the antecubital vein. Circulation times were six seconds (ether) arm-to-lung, and twelve seconds ($MgSO_4$) arm-to-tongue. Fluoroscopy showed a prominent pulmonary artery segment with displacement of the point of opposite pulsation downward and the esophagus posteriorly (on barium swallow). Left thoracentesis produced 1,500 c.c. on three different occasions. The fluid was pink, with many red blood cells microscopically. The specific gravity was 1.008. Two cell blocks for microscopic examination were reported as negative for malignant cells.

The clinical impression was rheumatic mitral stenosis, severe, and aortic stenosis, mild, with complicating hemoptysis. However, because of the sanguineous nature of the pleural fluid, gross hemoptysis, and wheezes, bronchoscopy was done. A friable, easily bleeding mass with narrowing and fixation of the left main bronchus was reported. Biopsy of the mass was reported microscopically as an oat-cell carcinoma.

Because of these findings, a left pneumonectomy seemed indicated, but acute post-operative right heart failure was feared because of the likely aggravation of the pulmonary hypertension seen with severe mitral stenosis. However, the possibility of a preceding valvulotomy to lessen the pulmonary artery pressure and possibly to accommodate the anticipated rise resultant from pneumonectomy was thought feasible.

Pulmonary function studies (Table I) were relatively normal. It was decided to evaluate the cardiac status and pulmonary hemodynamics by occlusion of one pulmonary artery as a measure of the rise that would have to be tolerated following resection. This was made possible preoperatively by the method first described by Carlens and associates.[4]



A. *B.*

Fig. 3.—*A*, Catheter in right pulmonary artery with balloon inflated. *B*, Angiogram of pulmonary artery to right upper lobe.

Cardiac catheterization was done. Moderate pulmonary hypertension at rest was found (Fig. 2). The pulmonary wedge pressure was elevated. The balloon was then inflated in the right pulmonary artery with 10 c.c. of 35 per cent Urokon (Fig. 3,*A*). The distal pressure dropped to the previously recorded pulmonary wedge value and the proximal pressure rose 13 mm. Hg systolic and 6 mm. Hg mean. Exercise resulted in a marked rise in distal and proximal pressures. The unoccluded exercise pressure was not determined. An angiogram of the right upper lobe pulmonary artery was obtained during occlusion with approximately 5 c.c. of 35 per cent Urokon (Fig. 3,*B*). The essential data are presented in Fig. 4. These tests were interpreted as showing enough ventilatory and circulatory reserve to permit surgery. The patient, however, developed a right upper lobe "pneumonitis" which delayed this.

On June 11, 1954, a thoracotomy was performed by Dr. George Holmes, who found no evidence of neoplasm. Dr. E. Fell then proceeded to open the finger-tip mitral orifice to a two-finger size. The left atrial pressure fell from 54 cm. saline (39 mm. Hg) to 44 cm. saline (32 mm. Hg).

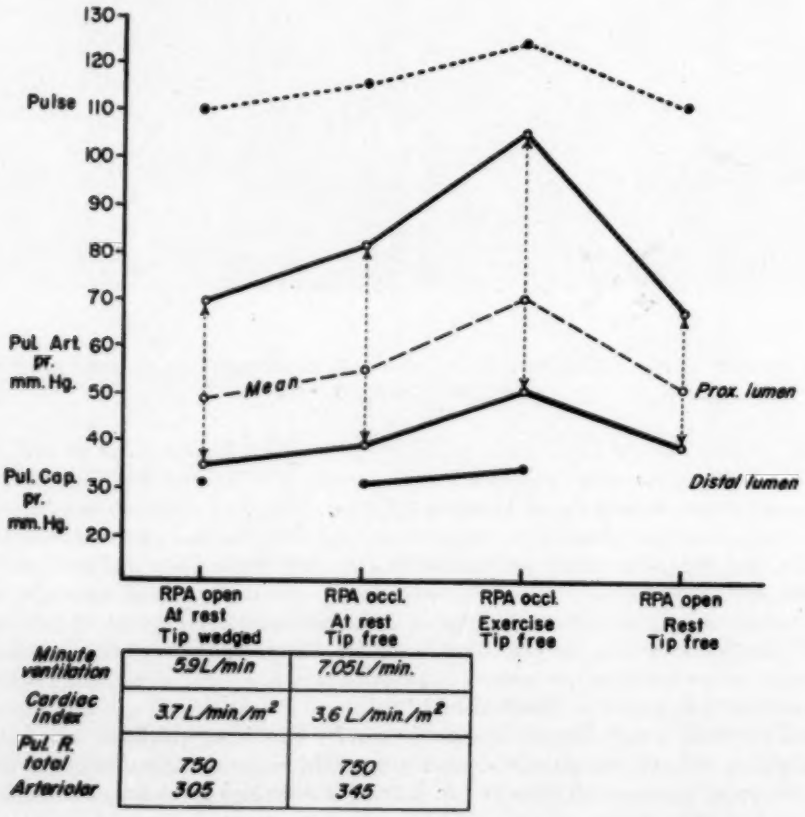
The postoperative course was marred by a mixed supraventricular arrhythmia, which responded to quinidine, and a thrombophlebitis at an intravenous cutdown site on the right leg.

TABLE I. PULMONARY FUNCTION

	OBSERVED	PREDICTED	PER CENT PREDICTED
Respiratory rate per minute	20/min.		
Tidal volume	430 c.c.		
Inspiratory reserve volume	1,350 c.c.		
Expiratory reserve volume	1,750 c.c.	1,320 c.c.	
Inspiratory capacity	1,780 c.c.	3,400 c.c.	
Vital capacity	3,450 c.c.	4,300 c.c.	81.5
Minute ventilation	6,100 c.c.		
Walking ventilation	11,850 c.c.	12,000 c.c.	
Maximum breathing capacity	96,300 c.c.	108,000 c.c.	89
Breathing reserve	93.5%	90 to 95%	
Walking index	12.5%	10 to 20%	
Air velocity index	1.09	0.9 to 1.0	
Minute oxygen consumption	187 c.c./min.		

Fig. 4.—Summary of catheterization results.

The pathologic slides have been reviewed and show no evidence of tumor. The patient has been subsequently rebronchoscoped, with a normal tracheobronchial tree reported. Hemoptysis has not recurred, and x-ray examination showed almost complete clearing of the pleural fluid and the right upper lobe lesion. The patient is now, six months postoperatively, gainfully employed for the first time in about one year.



COMMENT

We are presenting this case as tentatively supporting the concept that a patient with mitral stenosis can withstand major resectional pulmonary surgery in spite of the pulmonary hemodynamics involved.[7] It has been shown that normal dogs, following pneumonectomy, show only a mild rise in the pulmonary artery pressure at rest.[8] Also, it has been shown that patients with pulmonary disease and mild pulmonary hypertension often can withstand surgical removal of one or more lobes without a significant further rise in pressures.[9] There have been reports of good clinical results following lobectomy in patients with mitral stenosis.[10,11]

The balloon technique of Carlens presents a method of simulating pneumonectomy preoperatively. Results with this method have been reported by Hanson[5] and Nemir.[12] Previous experience with the method and studies of more than twenty-five cases will be published elsewhere.[13] The occlusion of the artery on the side of pneumonectomy, of course, would be the most conclusive test. However, the occlusion of the contralateral artery should give approximately the same result, the pressure rise being possibly a little higher if the artery of the affected lung has less reserve. The correlation of the distal pressure with the pulmonary wedge pressure may permit use of this as an alternate method of determining the pulmonary capillary pressure, especially when wedging is unsuccessful. It may actually be superior to wedging, since it eliminates the many factors which make wedge pressures somewhat uncertain. Occlusion was effected for over one hour with no subjective symptoms. However, the postcatheterization right upper lobe "pneumonitis" may represent a vascular insult due to stasis of the dye in the vessels. This is the first such complication to be reported. Electrocardiogram changes were not noted in this case or in the previously reported cases.[14]

In mitral stenosis, the pulmonary hypertension so often found is due to: (1) a mechanical obstruction at the mitral valve and (2) an increased vascular resistance of a labile and fixed character.[15,16] The mechanical obstruction is surgically remediable by adequate valvulotomy with a resultant immediate drop in the pulmonary artery pressure of the same magnitude as the left atrial pressure drop.[17,18] The vascular resistance responds (1) slowly, over a matter of months, with a further drop in pulmonary pressure,[19,20] and (2) to sleep[21] and various drugs (sodium nitrite, TEAB, Dibenamine,[21] Aminophylline,[22] Priscoline,[23] and hexamethonium),[24] with a fall in pressure but without a fall in blood flow. A residual component of a fixed, organic nature probably persists in severe cases, even after valvulotomy. No way of predicting this fixed component quantitatively has been developed. However, the preceding pharmacologic responses may offer a way.

The obstructing of one pulmonary artery produces a unique circumstance. Doubling the flow through the lung by mild exercise results concomitantly in a relative increase in the mitral valvular obstruction with an increased left atrial pressure. Any pulmonary artery pressure changes then may be related to this pressure change and/or to cerebral neurogenic factors of exercise. However, with the balloon obstructing one pulmonary artery, the contralateral blood flow

doubles, but the mitral flow and the cerebral factor should remain unchanged. Any changes then can be better evaluated. In this case, the resistance in the open lung halved itself, showing that the resistance was not fixed in at least one-half its usual value. Also, it offers further evidence for a reflex factor in the hypertension of mitral stenosis. The pharmacodynamic responses, furthermore, suggest the possible use of such drugs in the surgery for hypertensive patent ducti with reversal of flow. The acute pulmonary pressure rise expected after closing the ductus may be buffered or prevented by these drugs until the more delayed changes in vasculature can take place.

Another avenue of investigation which the balloon catheter technique[3] offers is its use during bronchospirometry to determine complete individual pulmonary functions. This might help to elucidate those cases where bronchospirometry shows that the oxygen uptake occurs predominantly in the lung with the poorer ventilation. However, the increase of blood through the open lung will change the normal perfusion-diffusion relationships.

In conclusion, we feel that when these two surgical procedures (valvulotomy and pulmonary resection) are indicated, the commissurotomy, if done first, will buffer, by the immediate drop in pressure, any rise that resection might eventuate. Until more extensive data are available, however, we believe that resectional tolerance determination is helpful. This may be done by the balloon technique preoperatively, with an indwelling catheter in the pulmonary artery, or by needle puncture with a strain gauge during surgery. Even if the pulmonary lesion is inoperable, commissurotomy would seem desirable to provide a better convalescence[25] and psychic support postoperatively.

SUMMARY

1. Pulmonary resection with mitral stenosis is discussed and its tolerability determined.

2. The nature of pulmonary hypertension is discussed.

3. The results of obstructing one pulmonary artery in a patient with mitral stenosis are presented.

SUMMARIO IN INTERLINGUA

Es reportate un caso que pareva presentar le problema de pneumonectomia pro carcinoma, con le existentia de hypertension pulmonar debite a stenosis mitral.

Es presentate le resultatos del pre-operative application del technica a catheter ballonate, destinate a simular le effecto de resection pulmonar. Per medio del uso de un ballon inflationate con aperturas proximal e distal il esseva possibile obtener significative datos hemodynamic. Il pareva exister un exacte correlation inter le pression "pulmono-capillari" obtenite per medios conventional e le pression registrate ab le apertura distal del ballon.

Le combination del prolongate obstruction de un pulmone con le injection de substantia de contrasto in le mesme pulmone resultava in un lesion pulmonar non previemente observate.

Le natura de hypertension pulmonar es discutite, insimul con un referentia al varie agentes usate in tests del elemento labile. Es listate varie situationes in que le technica a catheter ballonate pare utilisabile pro evalutar le function cardiopulmonar.

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DISTRIBUTION OF FLOW THROUGH A PULMONARY MANIFOLD

SIMON RODBARD, M.D., PH.D.*

CHICAGO, ILL.

THE lung requires a large volume in order to provide a surface area adequate for gas exchange. This space requirement, combined with the effects of the laws governing diffusion across membranes, produces certain physical and physiologic conditions which can markedly affect the distribution not only of the pulmonary blood flow but also of the loci of parenchymal and vascular disturbances in the lesser circulation. The arteries and veins of the lung are surrounded and supported by the parenchyma of the lung. The capillaries, however, are virtually suspended in the air spaces of the alveoli and receive no tissue or fluid support whatever; otherwise their respiratory function would be seriously embarrassed.

These conditions derive from the fact that the process of diffusion of oxygen and carbon dioxide across the alveolar capillary wall depends not only upon the differences in the partial pressures of the gases on the two sides of the capillary surface, but also upon the thickness of the respiratory membrane. Respiratory gas exchange is made possible by an extremely thin capillary membrane between blood stream and alveolar gas.[1] A highly efficient arrangement of this type is present in the lungs since the thickness of the alveolar capillary-epithelial membrane separating blood from air is only a few microns in thickness.[2]

If the blood pressure in the capillary were raised above the osmotic pull of the plasma proteins, transudation of the fluid across the gossamer capillary wall would result. The functional thickness of the wall would then be increased by the added film of fluid, and the efficiency of the lung as a respiratory organ would be reduced.

In the normal human lung, the height from apex to base may be 30 cm., while the anteroposterior diameter may be 20 cm. in depth.[3] If its blood vessels were homogeneously filled with fluid, a passive hydrostatic pressure of about 23 mm. Hg would be present in the basal capillaries in an upright human lung. With the further addition of a normal pulmonary arterial pressure, the total force acting in the dependent pulmonary vessels would be raised to about 35 mm. Hg, sufficient to overcome the osmotic pull of the blood, and transudation with

From the Cardiovascular Department, Medical Research Institute, Michael Reese Hospital, Chicago, Ill.

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*Present address: University of Buffalo School of Medicine, Chronic Disease Research Institute, Buffalo 14, N. Y.

pulmonary edema would become inevitable in the dependent portions. Further, the gravitational effects might affect the distribution of flow through the various portions of the lung, and in this way lead to pulmonary vascular-ventilatory dysfunction.

The problems posed by the foregoing hydraulic considerations were approached by simulating some of the dynamics of the pulmonary circulation in specially constructed models.

In the present experiments, in vitro models designed to simulate certain aspects of the lung were used. To assist in comparison with the presumed state of affairs in the lungs, however, clinical terminology has been used interchangeably with mechanical terminology. For example, terms such as apex and base of the lung, emphysema, pulmonary venous congestion, and other anatomic and clinical nomenclature are used, with full knowledge that these refer directly to the simulated situations and less certainly to the actual state of affairs in the functioning lung. The results obtained with the model and the insight into certain problems of cardiopulmonary physiology appear to justify this approach.

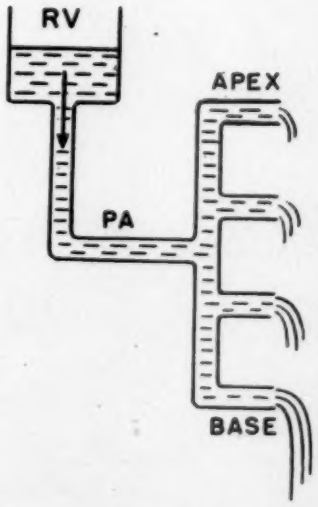

Fig. 1.—The rudiments of the pulmonary model. *RV* represents the hanging reservoir maintained at several predetermined levels, serving as a "right ventricle." *PA* represents the pulmonary artery. Flow is distributed to four tubes (arterioles) and the delivery through each is measured.

METHODS

The basic model is shown diagrammatically in Fig. 1. A hanging reservoir simulating the right ventricle (*RV*) provided a constant pressure head. A rotameter (not shown) between this reservoir and the manifold gave a measure of the total flow. Fluid passed from the reservoir (*RV*) through a wide tube (*PA*) into a manifold which divided it into four smaller tubes (arterioles) ar-

ranged at a distance of 10 cm. from each other. Since a variety of arrangements was utilized to test specific effects, the particular methods for each set of experiments are given with the results of that series.

RESULTS

Two general types of experiments were done. In the first group the dynamics of flow through a rigid system was studied. Against the background of these data, the effects of the introduction of soft-walled "capillary" tubes were determined.

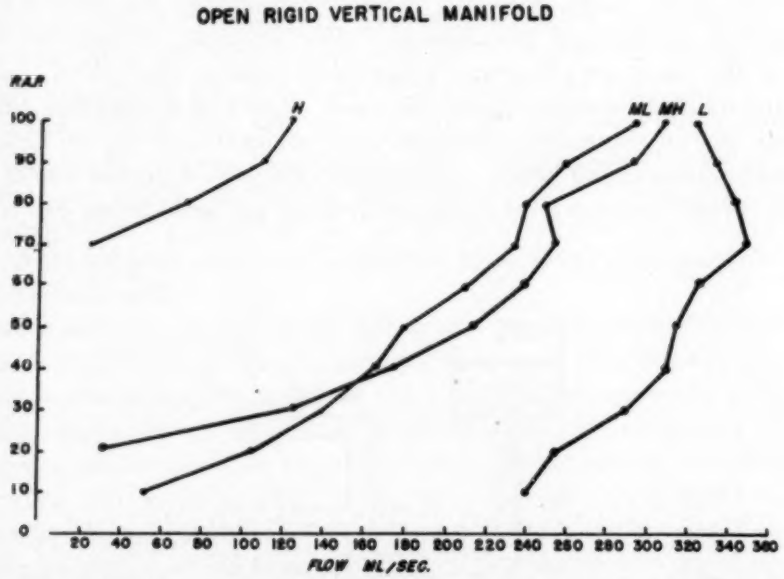

Fig. 2.—Delivery through the manifold in Fig. 1, with *vertical* positioning. The lowest outflow tube (*L*) received the greatest flow; the mid-low (*ML*) and mid-high (*MH*) tubes received significantly less; the highest tube received practically no flow until a pressure head of 70 cm. water was used. Discussed in text.

A. Rigid Tubes.—

1. Division of flow through four separate outlets: The flow from the main vessel (PA) was divided into four separate outlet channels, 10 cm. apart, by causing it to pass into a set of Y tubes which divided the flow first into two streams, and thence into four streams by means of a second cascade of two Y tubes. The outflow from each of the four tubes was collected in graduated cylinders and the delivery per unit of time was measured.

The four outlet tubes were first placed on the same horizontal level. Under these circumstances the delivery through each of the four outlets was approximately, but not exactly equal. Each outlet received approximately 25 per cent ± 3 per cent of the total delivery. The variation from equality is due to the complex interrelationships introduced by the angle of deviation of stream lines, and the initiation of patterns of secondary flow.[4] However, the flow through each of the four outlets was sufficiently similar at most pressure levels to provide a satisfactory base line for further study.

2. The effects of vertical position: The four outlets were now placed in a vertical position, arranged one above another at distances approximately 10 cm. apart. The uppermost tube was labeled the "high" (*H*) tube, and the lowest tube was labelled as the "low" (*L*) tube. The intermediate tubes were called "mid-high" (*MH*) and "mid-low" (*ML*), respectively. As expected, outflow took place primarily through the lower outlets (Fig. 2).

The height of the pulmonary arterial pressure also affected the distribution of flow through the four outlets. At very low arterial pressures, most of the flow passed through the low channel while virtually none took place through the high channel. The intermediate tubes shared in this distribution to a smaller extent, these flows also being divided on the basis of arterial pressure and altitude of the segment.

As the pressure head was raised, flow through all the tubes increased progressively. At a pulmonary arterial pressure of 20 cm. water, the flow through the L tube was two-thirds of the total delivery, with smaller amounts passing through ML and MH. Virtually no flow passed through H until the pulmonary arterial pressure was raised to significantly higher levels, about 70 cm. water or more. With still higher arterial pressures, flow through the high tube of the vertical manifold increased rapidly.

These data served to emphasize the important potential effects of gravity on flow distribution through a simple vertical manifold.

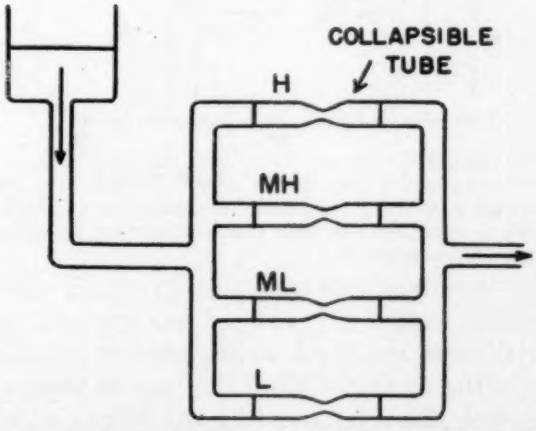

Fig. 3.—A manifold with intermediate ("capillary") portions consisting of soft-walled (Penrose) tubing. The outlets for each of the four tubes are at the same level.

3. Uniform outlet level: The pulmonary system in animals and man differs from the arrangement utilized in section 2 in that the outlet (pulmonary vein) is at approximately the same level as the inlet (main pulmonary artery). To test the effect of the outlet level on the distribution of flow, wide-bore rigid tubing was attached to each of the tubes so that the outlets of all four tubes were brought to the same horizontal level. The essential features of this arrangement are given in Fig. 3, except that the collapsible tubes noted are replaced

by rigid tubes of uniform diameter. Under these circumstances, flow through all four tubes (capillaries) became approximately equal with flow distributions essentially similar to those obtained with an open *horizontal* manifold as in section 1 above.

These results differed from those described in the immediately preceding sections since delivery in a system of rigid pipes is a function primarily of the difference between the inlet pressure and the outlet level. For tubes that rise a given distance above the final outlet, as in the high and mid-high tubes, the resistance offered by this rise is counterbalanced by a siphonage effect produced

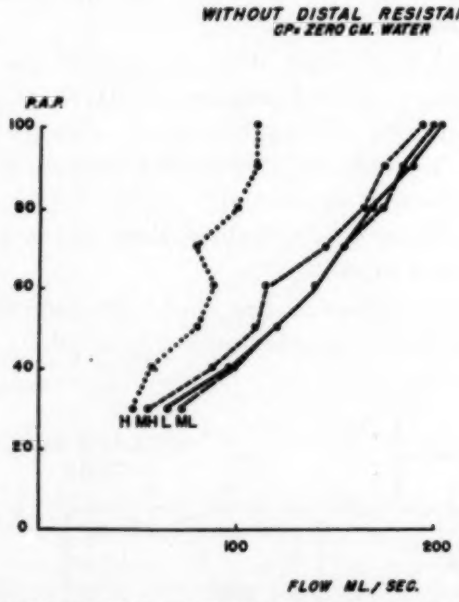

Fig. 4.—Delivery through a manifold containing "capillaries" as in Fig. 3, vertical position. Flow through the high (*H*) tube is definitely less than that through the lower tubes, especially at higher pulmonary arterial pressures (vertical scale).

in the tube which returns the fluid to the common outlet level. Similarly, segments below the outlet as in the basal (L) vessels have a higher driving pressure acting on them, but this is balanced by the height which must be traversed before the fluid attains the common outlet. The distending (lateral) pressure in the more dependent tubes is greater than that in the higher tubes; however, in a rigid system this has no effect on flow. By contrast, these differences in altitude can produce marked effects on flow in a system of soft-walled tubes, such as are present in the body.

B. Soft-Walled "Capillaries."—

In this set of experiments each rigid brass tube which had served as a "capillary" in the section above was replaced by a segment of soft-walled surgical Penrose tubing 3 cm. long and 6 mm. in diameter (Fig. 3). The outlets of all

four tubes were placed at the same level, equal to that of the inflow. In actual practice a separate outflow was available for each of the levels (*H*, *MH*, *ML*, and *L*), but this separation is not shown in the figure.

Horizontal position: With the manifold in the horizontal position, flow through each of the four tubes was approximately equivalent. These effects may be expected since the conditions of flow and resistance in each horizontal tube are approximately equal to those of the other tubes of the same level. Some differences in flow were present but these were relatively small.

Vertical position: When the manifold was placed in the vertical position, notable changes in flow patterns were observed (Fig. 4). In the pressure range of 30 to 100 cm. of water the flow through each of the three lower tubes was approximately equal. The flow through the high tube was somewhat less than through each of the other three.

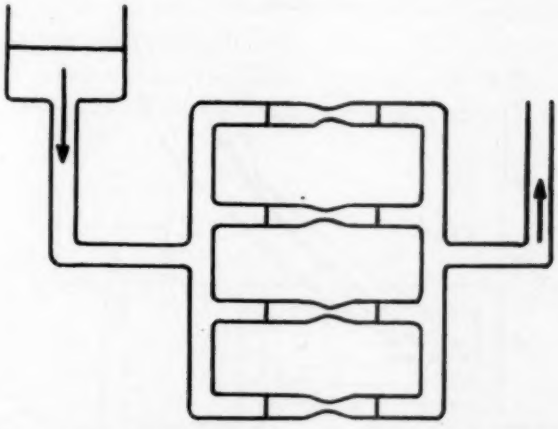

Fig. 5.—Manifold with outlet levels for all tubes at that of the high (*H*) tube. In the present arrangement this modification is equivalent to a rise in the "pulmonary venous" pressure of 15 cm. water (11 mm. Hg).

Observation of the system during flow demonstrated that, compared with the distended condition of the lower tubes, the higher tubes were partially collapsed. These differences in diameters could account for the reduced flow through the higher channels.

As the pulmonary arterial pressure was raised, flow through all four outlet systems was increased progressively. The absolute increase in flow through the low segments was in general greater than that through the higher tubes (Fig. 4).

Comment: The redistribution of flow is probably due primarily to the differences in diameter of the soft-walled tubes, in accordance with Poiseuille's law. At the base of the manifold the soft-walled tube is distended by the pressure head as well as by the resistance provided by the outlet head, equal to the height from the basal (L) capillary to the outlet (pulmonary venous) level. The diameter of L is distended and a higher rate of flow is obtained through this part.

By contrast, the column of fluid from the high tube to the level of the pulmonary vein produces a negative pressure (siphonage) effect sufficient to collapse this vessel; its effective diameter is reduced, enhancing the resistance to flow through it. At very low driving heads in the pulmonary artery, the pressure is not sufficient to drive fluid to the apex of such a vertical "lung" and this portion therefore receives little or no delivery. With higher pulmonary pressures, delivery to the apex takes place, but at significantly reduced flow, compared to that passing through the base.

Outlet (distal) resistance: If the outlet (pulmonary vein) pressure level is equal to that of the apex, the siphonage effect and the consequent collapse of the high vessels are diminished and eliminated. To test the effect of this arrangement on flow the outlets of each of the four tubes were placed at the level of the high tube (Fig. 5).

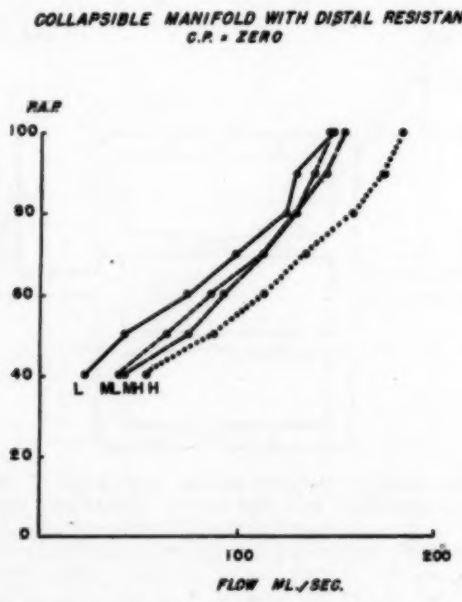

Fig. 6.—Flow through the manifold illustrated in Fig. 5, vertical position. The outlet level of all four channels is at the height of the high tube. Conventions as in Fig. 2.

Under these conditions, the total flow was decreased somewhat, but delivery through the apical (*H*) tube was significantly enhanced (Fig. 6). Increases in the driving (*PA*) head from 50 to 100 cm. water produced essentially the same order of rises in flow through each of the four outlet systems.

These results provided further evidence that vertical placement of the tubes produced its effects in part by virtue of the fact that the higher tubes tended to collapse because of siphonage. The introduction of a distal resistance eliminated the siphonage collapse of the high tube and thereby approximately equalized flow through all channels.

Intrapulmonary air pressure: In the course of respiratory activity, small but significant pressure changes take place in the airways and in the alveoli of the lung. During inspiration the intrapulmonary air pressure falls a few

centimeters of water and dilatation of the airways permits a relatively free entry of air into the respiratory spaces. During expiration the intrapulmonary air pressure rises somewhat since in this phase the reduced caliber of the airways increases the resistance to outflow of air from the lungs.

These effects may conceivably play significant roles in the distribution of flow through the soft-walled pulmonary capillaries. It is apparent that a heightened intrapulmonary pressure, particularly during expiration, would compress such delicate vessels and thereby increase the resistance to flow through them.[5] In certain conditions in which bronchospasm or inflammatory processes produce entrapment of air in the respiratory passages, rather high air pressures in the lung may result, and more dramatic effects on flow distribution may be produced.

In order to simulate this compression of capillaries by intrapulmonary air pressures, each of the four soft tubes of the model was enclosed in a glass cylinder (Fig. 7). Pumping of air into these cylinders increased the "alveolar" air pressure surrounding the soft "capillary" tubes. The effect of various degrees of "intra-alveolar" pressure on the flow through the four tubes was thereby investigated. In this series, the outlet (PV) was at the same level as the inlet (PA).

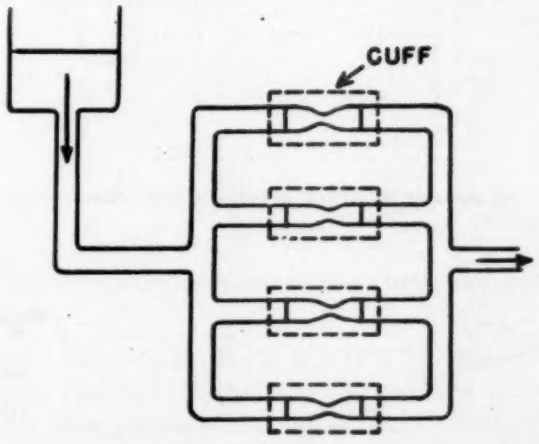

Fig. 7.—Schema for increasing the pressure outside the "capillary" vessel wall, simulating an emphysematoid state. Each soft-walled tube is enclosed in a glass cylinder. The air pressure (CP) is set at predetermined levels and is uniform in all four channels. All four tubes in this experiment emptied at the level of the inlet.

When the manifold was in the *horizontal* position, an increase in the alveolar pressure affected flow in all four tubes to an equal extent. As the cuff pressure was increased the tubes were narrowed progressively and there were reductions in flow through all of the tubes. These effects were of the same order as that produced by applying pressure against the outer wall of a single soft tube.[7]

In the *vertical* position, a uniformly distributed increase in cuff (alveolar) pressure on the four capillaries resulted in a marked augmentation of flow through the basal tubes at the expense of flow through the apical tubes.

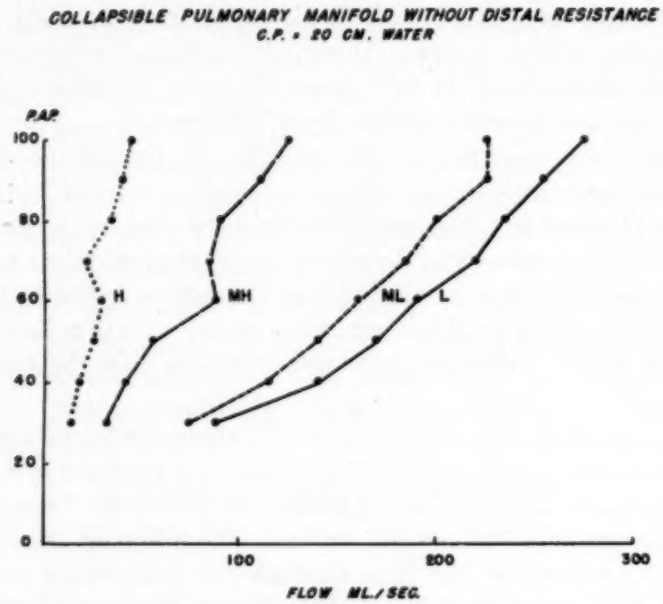

Fig. 8.—Flow through a manifold as described in Fig. 7, vertical position. The air pressure in the cylinder was maintained at 20 cm. water. The vertical axis gives the various "pulmonary arterial" pressures used; the horizontal axis gives the flow in milliliters per seconds. Conventions as in Fig. 2. Discussed in text.

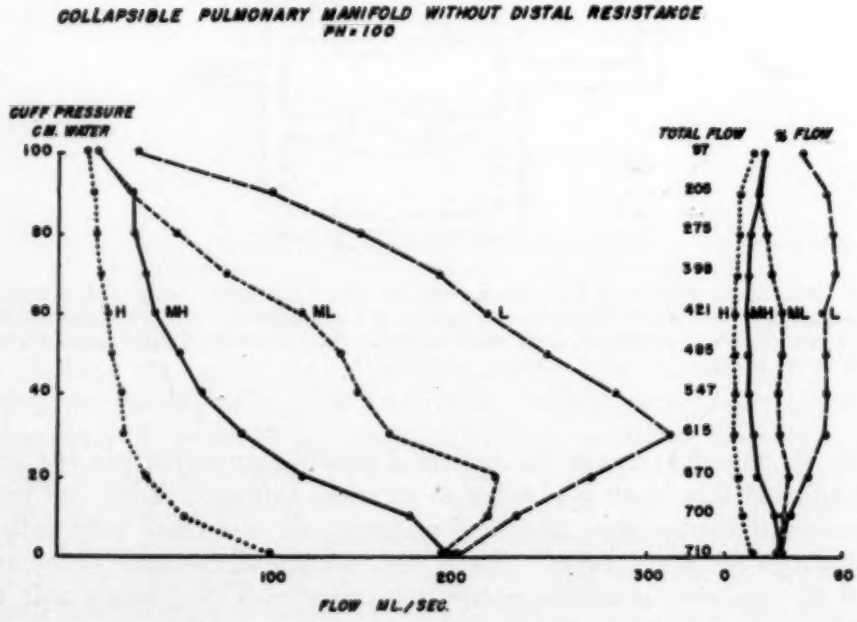

Fig. 9.—Effect of cuff (intrapulmonary) air pressure on flow. The vertical axis gives the settings of the cuff pressure; the horizontal axis gives delivery. The total flow (milliliters per second) at each cuff pressure is given in the column to the mid-right of the figure. The percentage distribution of this flow through each of the four channels is given at the right.

By comparing Fig. 8 with Fig. 4 a representation of the effect of this cuff pressure can be obtained. At zero air pressure (Fig. 4), flow through the three lower tubes was nearly equal, while that through the high tube was somewhat reduced. The imposition of a uniform air pressure of 20 cm. water produced an enhancement of flow through the low tubes, while flow through the higher tubes was reduced (Fig. 8). Increases in pulmonary arterial pressure raised the flow through all tubes approximately in equal proportions, but the absolute difference in flow became more marked. At an arterial pressure of 100 cm. water, delivery through the low tube was nearly ten times that of the high tube.

Generally, similar effects were produced at other air pressures. The effects of various air pressures on flow are shown in Fig. 9, in which all values are for an arterial pressure head of 100 cm. water. It can be seen that as the air pressure is raised to values of about 30 cm. water, flow through apical (*H*) and mid-high (*MH*) tubes falls off sharply. The flow through the basilar tubes (*ML* and *L*) increases until the cuff pressure is 30 or 40 cm. water. At 30 cm. air pressure, for example, the flow through the dependent half of the manifold is more than five times that through the upper half. With further increases in air pressure, the flow in all tubes of the manifold decreases progressively and the effect of the altitude of a given tube is reduced accordingly. This is illustrated in the section on the right of Fig. 9 which gives the percentage distribution of flow through each of the four tubes.

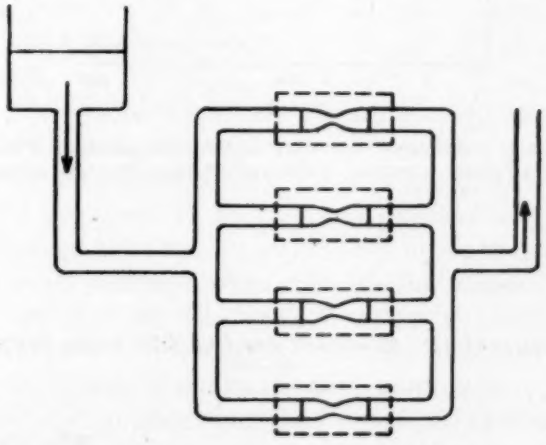

Fig. 10.—This schema represents the combined effects of a distal resistance and of cuff pressure.

These studies suggest an important role for intrapulmonary air pressure on the distribution of blood flow through the various levels of the manifold.

Intrapulmonary air pressure plus enhanced distal resistance: In certain conditions, a heightened pulmonary venous pressure may be present simultaneously with an increase in intrapulmonary air pressure. To obtain data on the effects of this combination on flow distribution through the manifold, the arrangements given immediately above were utilized, except that the outlets from all of the four tubes of the manifold were elevated to the level of the high tube (15 cm. above the "normal" outlet) (Fig. 10). It has already been noted

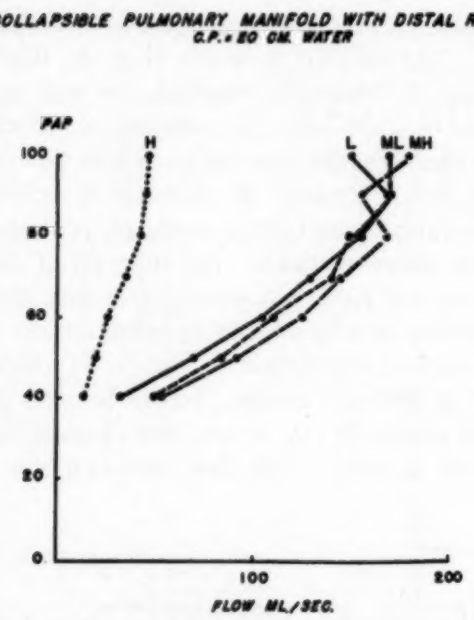

Fig. 11.—This graph is constructed according to the arrangements in Fig. 8. It differs in that it represents the effect of an outlet resistance (pulmonary venous pressure) of 15 cm. water as in Fig. 6. Discussed in text.

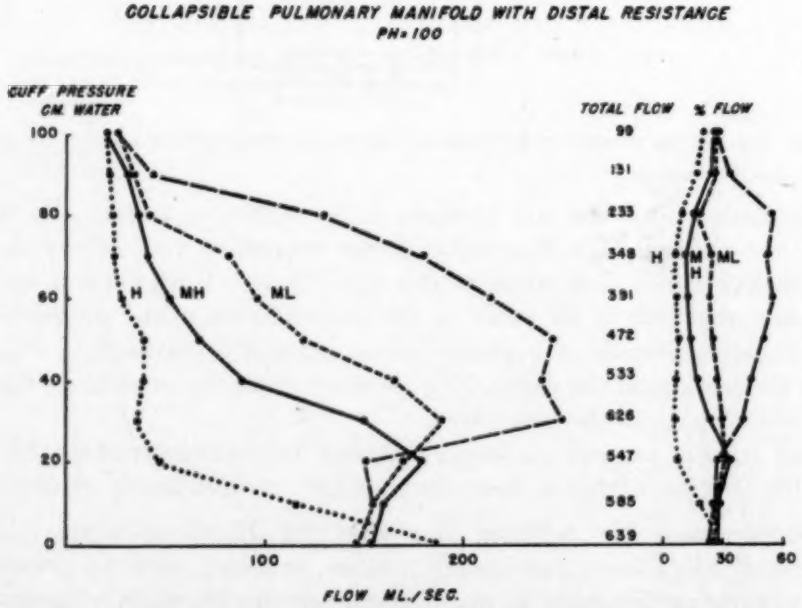

Fig. 12.—Effect of cuff pressure on delivery in the presence of an outlet resistance. The data are given for a driving pressure head of 100 cm. water. This graph is arranged similarly to and should be compared with Fig. 9. The distal resistance equalizes flow at low cuff pressures. As cuff pressure is increased, flow though the upper segments of the manifold is reduced to a greater extent than that through the lower segments. L flow is given by the unlabelled line at the right of each of the two graphs. Discussed in text.

that a distal resistance acts to increase delivery through the apex, making its flow equal to that through the other tubes of the manifold (Fig. 6).

In Fig. 11 the effects of a combined outlet (distal) resistance and an increase in cuff (intrapulmonary) air pressure of 20 cm. of water on the distribution of flow is given. The distal resistance tended to equalize flow through all parts of the manifold (Fig. 6). Further addition of air pressure reduced flow through the H tube, and increased flow through the lower remaining tubes (Fig. 11).

As the air pressure was raised progressively, flow through the apex began to decrease while flow through all the other channels increased somewhat (Fig. 12). At an air pressure of 30 cm. water, *L* flow increased rapidly at the expense of that through *H*; this increase was maintained as the cuff pressure was raised until it reached the value of 60 cm. water. Above this level, flow fell off rapidly in the low tubes as well as in the others. At cuff pressures of 90 or 100 cm. water, flow in all the tubes became minimal, but approximately equal.

Thus, while the presence of an outlet resistance tended to equalize flow through all the tubes of the manifold as previously noted, the application of air pressure eliminated this equalizing tendency. With an air pressure of 20 cm. water, the high tube received less flow than the other tubes and this preferential distribution held for all arterial pressures up to 100 cm. water.

The effects of various air pressures on flow distribution at an arterial pressure of 100 cm. water are given in Fig. 12. It should be noted that increases in air pressure in the range of 30 cm. water actually acted to decrease the over-all resistance, resulting in an enhanced flow through the manifold as a whole.

In rigid tubes, elevation of the outlet level has the effect of an equal reduction in the driving head, with a resultant decrease in delivery. In soft-walled tube systems, this relationship is obviously more complicated, and flow actually increases under certain circumstances as the outlet level is raised.[8]

The effect of a rise in the pulmonary venous pressure may thus produce anomalous increases in total delivery; the superimposition of "alveolar" pressure can bring about striking variations in the distribution of flow through a pulmonary system.

Precapillary resistance: In certain pathologic states the pulmonary arterioles may, as a result of excessive development of the intimal or medial coats, be transformed from the normal wide-diameter vessels to channels with a markedly reduced lumen. This occurs most commonly under conditions of persistent pulmonary hypertension, as in large ventricular septal defects, in aorto-pulmonary communications, and sometimes in mitral stenosis.[9,10]

The possibility that such changes in the smaller pulmonary vessels might affect the distribution of flow through the pulmonary system was investigated. To simulate an increased precapillary vascular resistance of this general type, the diameter of the "arterioles" of the model was reduced by introducing a 1 cm. long segment of glass tubing with a capillary lumen (2 mm. diameter) into the manifold just upstream from the capillary (Fig. 13).

Under these conditions of precapillary resistance, the total flow was reduced, but was distributed approximately equally through each of the four tubes (Fig. 14). This near-uniformity of flow distribution was apparent when the

driving head was 50 cm. water or more. For example, at a driving head of 100 cm. water, the flow through the low tube was 120 ml. per second, while that through the high tube was 98 ml. per second. This distribution is significantly different from that produced when no precapillary resistance was present (see Fig. 4).

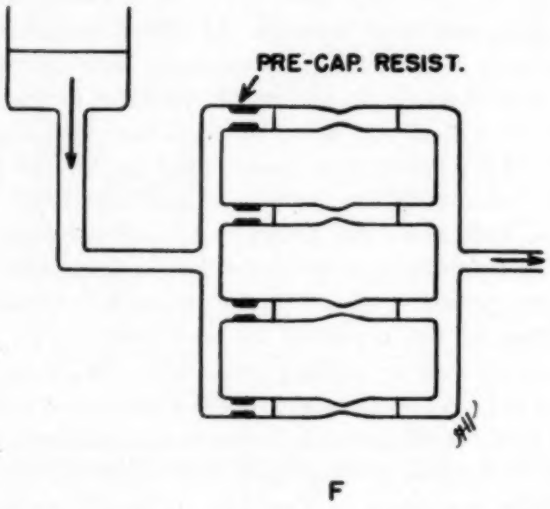

Fig. 13.—Schema of the pulmonary circulation with increased precapillary resistance. This is similar to Fig. 3, except that a piece of tubing with a small lumen indicated by the heavy black line was placed upstream to the elastic "capillary." Discussed in text.

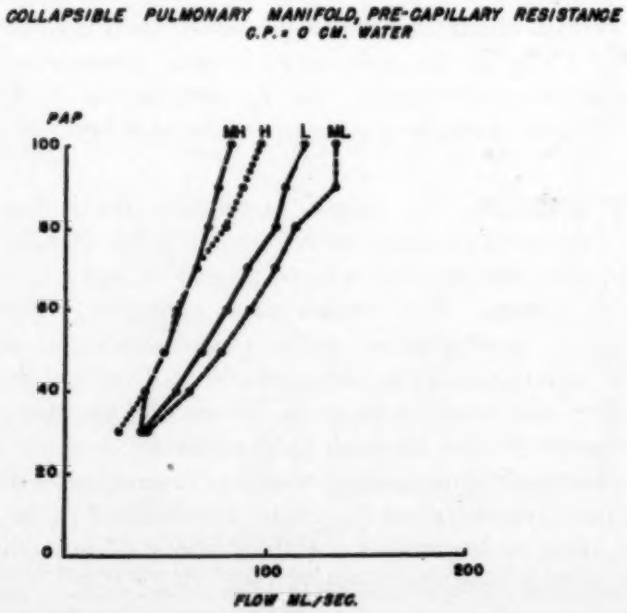

Fig. 14.—Flow through a vertical manifold with an increased resistance upstream to the capillaries. A similarity in flows through the four available channels is indicated for pulmonary arterial pressures up to 100 cm. water. However, the total flow is reduced below that given in the previous figures.

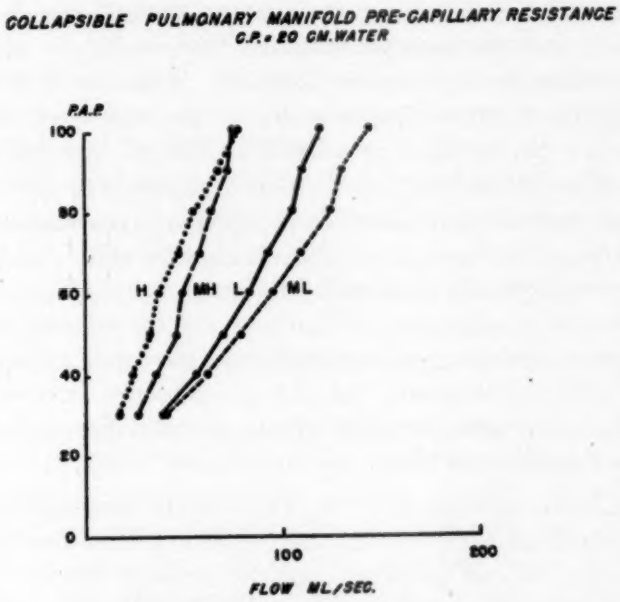

Fig. 15.—Delivery through a manifold as in Fig. 13 with a high precapillary resistance and a cuff pressure of 20 cm. water.

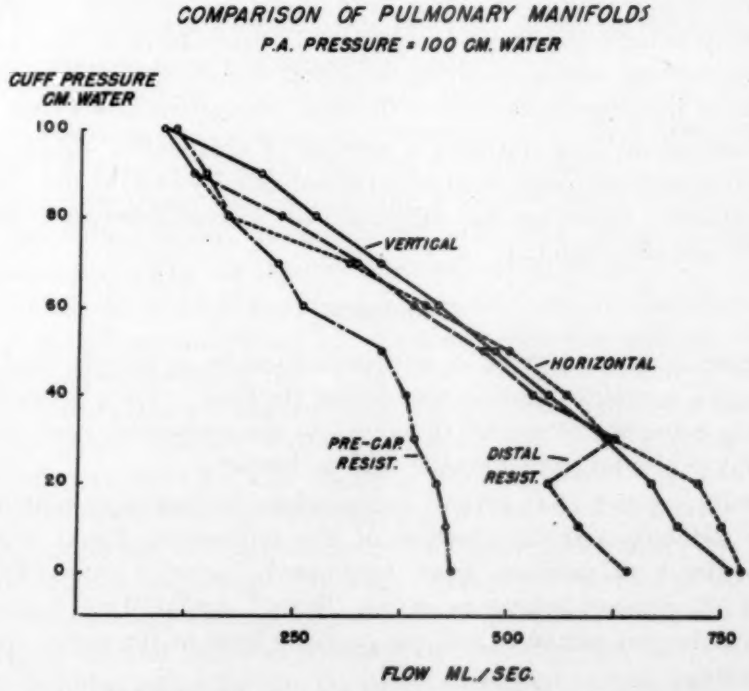

Fig. 16.—A comparison of the total flow through the manifolds used. The total flows are essentially of the same order for the vertical and horizontal positions. The addition of a distal resistance reduces flow. However, at cuff pressures of more than 20 cm. water, the flow equals that in the systems with a lower pressure difference between inlet and outlet. The precapillary resistance markedly reduces flow for all conditions except at high cuff pressures, at which the effect of the precapillary resistance becomes of less importance.

In the system with precapillary resistances, an increase in cuff pressure, by means of an air pressure arrangement like that in Fig. 7, affected the flow through the four tubes in an irregular fashion. Thus, at a pressure head of 100 cm. water, raising of the air pressure to 20 cm. water had no effect on the flow through the low tubes which remained at 120 ml. per second, while that through the high tube was reduced to 78 ml. per second (Fig. 15).

These results demonstrate that a precapillary resistance of significant degree eliminates most but not all of the differential effects of position on the flow distribution through the manifold. It must be noted that this effect is obtained at the cost of a reduction in total delivery for a given driving pressure head. Further, the normally great redistributing effects of increased pulmonary air pressures are greatly reduced. As the air pressure is raised, some redistributive effects may be seen, but the effects on total flow are not as striking as in the previous experiments cited.

Pulmonary vascular resistance: Inspection of the flow patterns through the manifold as a whole demonstrates that the position of the manifold, the degree of "alveolar pressure" or cuff pressure, and the pressure level at the outlet each can produce anomalous effects (Fig. 16). In general, it may be stated that elevation of the pulmonary arterial pressure increases total flow, while it will be reduced by a fixed precapillary resistance. However, the application of air pressure and in particular the elevation of outlet (pulmonary venous pressure) levels can sometimes produce apparently paradoxical increases in flow. This was particularly notable in the air pressure range from 20 to 30 cm. water (Fig. 16) where a procedure which might be expected to increase resistance actually led to significant increases in total flow through the system as a whole.

These features of flow through a system of soft-walled tubes may limit somewhat the significance and interpretation of calculations of the "pulmonary vascular resistance" based on the difference in pressures between the driving pressure head and the outlet.

DISCUSSION

The present studies serve as an approach to some of the physical problems of flow through a complex manifold simulating the lung. They provide a degree of insight into some of the special dynamics of the pulmonary vascular system which can play profound roles in health and in disease.

The results suggest that several independent factors may play important roles in the delivery and distribution of the pulmonary blood flow. These include the effects of pressure head (pulmonary arterial pressure), altitude (position) of the various segments of the "lung," precapillary (arteriolar) resistance, intra-alveolar pressure, and the pressure level at the outlet (pulmonary venous pressure).

When the pulmonary manifold is in a horizontal position with all segments at equal altitude, each of the separate vessels receives essentially an equal distribution of flow. The very nature of the lung structure, however, demands that some of its parts be above or below the inflow or outflow levels. In the adult,

lung tissue is present both 15 cm. above and 15 cm. below the main pulmonary artery and vein. A difference of 20 mm. Hg pressure may therefore be in operation between these two sites. Such pressure differences can produce important effects, particularly in view of the low pressures normally present in the pulmonary artery (25/10 mm. Hg or 34/14 cm. water).

Because of the collapsible nature of the thin-walled pulmonary capillaries, such an arrangement could enhance flow through the base at the expense of flow through the apex. The normal diastolic pressure in the pulmonary artery is therefore hardly sufficient to produce flow through the elevated apical vessels. Changes in the position of the body, however, such as occur in recumbency, can operate to redistribute the blood supply and to increase the flow to the apical segments.

This general concept has been the basis of one of many explanations[11,12] for the tendency to apical localization of reinfection tuberculosis in the adult, as well as for the recumbent therapy of this disease. This concept has had excellent support from the experimental work of Medlar and Sasano[13,14] and the pathologic findings in cattle. Rich[15] has reviewed other interpretations advanced to explain this phenomenon, including the role of lymphatics,[16] blood stream separation of the two venae cavae,[17] and gaseous exchange differences.[15] The present results provide a physical basis for the concept that the upright position may act to reduce the blood flow to the apex. Such changes in blood flow at the apices could form the basis for differences in the gas content in various segments of the lung[18,19] as well as in the rate of lymph drainage and in the distribution of blood from the two venae cavae.[20]

The higher incidence of tuberculosis reported in patients with pulmonic stenosis accords with the foregoing interpretation,[21,22] since the pulmonary arterial pressure available for perfusion of the lung is below normal.

The recumbent position, by bringing a greater blood supply to the apical segments, according to the viewpoints set forth, may be one of the important mechanisms operative in the therapy of apical tuberculosis.[23]

An increase in intrapulmonary air pressure usually decreased total flow, although it sometimes increased it. However, the distribution of blood flow through the various parts of the "lung" was markedly affected. The delivery through the basal portions was increased significantly, at least at low "alveolar" air pressures, while delivery through the apex was quickly reduced almost to vanishing proportions. In this regard it is of interest that emphysema and the rapid progression of apical tuberculosis often go hand in hand.

A progressive rise in intrapulmonary air pressure increases the impediment to flow through the lungs and ultimately demands a relative pulmonary hypertension if the systemic venous return is to be pumped through the lungs. As the pulmonary arterial pressure is raised an increased flow may be provided to the apices, but this enhances the tendency to basilar congestion, transudation, and edema. These tendencies may be countered by external positive pressure devices or by the development of a positive intrapulmonary pressure by the spontaneous air entrapment which occurs in emphysematoid respiration. The data obtained from the present experiments suggest, however, that the flow

through the apex may thereby be further reduced. If, however, the alveolar air pressure were raised excessively, the added load on the right heart might bring about its failure and lead to chronic cor pulmonale.

An increase in outlet (pulmonary venous) pressure markedly affects the flow distribution, with the production of a notable increase in flow through the apical vessels. Thus, the data suggest that in pulmonary venous hypertension such as may occur in stenosis or insufficiency of the mitral valve or in left heart failure due to any cause, the flow through the apex may be enhanced. It is perhaps significant that the incidence of apical tuberculosis is statistically less frequent in patients with mitral stenosis.[25]

An interesting facet of the effects of a raised outlet (pulmonary venous) pressure has to do with the fact that a significant increase in flow may result, despite a reduction in the effective pressure head driving fluid through the lungs. This effect appears to result from an increased distention (diameter) of the elevated vessels, thereby permitting an increased flow through these parts. Calculations of the total pulmonary vascular resistance on the basis of the difference between the pulmonary arterial and the outlet pressures may thus be complicated somewhat. Comparison of Figs. 9 and 12 demonstrates more flow under certain conditions in the presence of an outlet resistance than when there is no such outlet resistance.

The rise in outlet (PV) pressure produces other effects. Thus, the increase in the lateral pressure on all the pulmonary capillaries may act to bring the dependent capillaries closer to the threshold of transudation and edema. This effect may be counteracted, as above, by an increase in alveolar air pressure.

A distinctive character is evident in the pulmonary vascular hypertension produced by a heightened resistance at the outlet. Thus, all elements of the pulmonary system are involved in such a generalized pulmonary hypertension: outflow tract, capillaries, and arterial ramifications; the engorged capillaries lead to the danger of pulmonary edema. By contrast, pulmonary arterial hypertension, as occurs in emphysema and in some congenital anomalies, may affect only the pulmonary arterial tree, but the outflow tract and capillary pressure may be unaffected, and there is no threat of pulmonary congestion or edema.

If in the presence of an enhanced outlet resistance the pulmonary air pressure is increased uniformly, flow through the apex decreases rapidly while that through the base may even increase. This is an effect essentially similar to that without an outlet resistance. This suggests that while an elevated pulmonary venous congestion might increase apical blood flow, a compensatory air entrapment would tend to reverse this effect and reduce the flow through these elevated regions.

The assumption of an upright posture in such instances, as in orthopnea, could therefore make the relatively uncongested elevated portions of the lung available for aeration although a limited flow would course through these elevated levels. The result would be an improvement over the previous respiratory situation but would still be a relatively inefficient type of response. A complex cardiopulmonary relationship seriously embrassing the mechanisms adjusting pulmonary blood flow may result.[6]

Data are accumulating to show that unequal air distributions and air entrapment may occur not only in emphysema but also in left heart failure and even in normal subjects. Evidence for the differential perfusion of elevated as against dependent parts of the lung has been obtained recently by studies using bronchospirometry or the introduction of fine catheters into the bronchi.[18,19] The concentrations of oxygen and carbon dioxide in the elevated portions of lungs of normal subjects suggest a reduced flow through these parts, compared with the more dependent portions of the lung. Segmental air entrapment and pulmonary arterial vasoconstriction may play a role in the adjustments, but certainly gravitational factors can account for much of this effect. This is emphasized by our findings that even slight air entrapment can virtually stop flow through an elevated segment while flow through a dependent segment is hardly affected by an equivalent "intra-alveolar" pressure.[26]

These considerations suggest that in clinical pulmonary congestion the restlessness and frequent change of position may be a protective response which serves to alternate the relative altitude of various portions of the lung and in this way perhaps intermittently relieve, and exchange, regions of congestion and edema. The hypostatic pulmonary congestion and pneumonia of unconscious patients probably results from the loss of just such a protective mechanism.

When the pulmonary arteries have been exposed for a sufficient period to a heightened tension, pathologic changes may occur in the vascular network of the lesser circulation.[9,10] In the smaller vessels these changes may lead to a reduction in the lumen of the arterioles. Such an eventuality would reduce the blood pressure acting on the capillaries but by the same token it would demand a further increase in pulmonary arterial pressure, and thereby place an added strain on the right heart. The results suggest, however, that the interposition of a precapillary resistance between the right heart and the pulmonary capillaries can achieve a relatively uniform distribution of the blood flow through the lung, thus improving ventilatory-respiratory function. Indirect evidence for such an improvement has already been presented.[27]

The redistributive effects of intrapulmonary air pressure on flow distribution are reduced in the presence of a heightened precapillary resistance, and flow through the elevated portions of the lung is not diminished as drastically as when the pulmonary arterioles provide little resistance to flow. The benefits obtained in terms of protection of the pulmonary capillary against transudation and in the relatively uniform distribution of pulmonary blood flow are thus paid out of the increased work load of the right ventricle which is called upon to generate a higher arterial pressure to pump away its venous return.

The blood supply of the parenchymal tissues of the lung, being derived from the systemic bronchial arteries, probably does not share directly in the potential types of distribution of the pulmonary arterial blood flow previously discussed. This may be assumed on the basis of the higher blood pressure level in this system and the fact that the bronchial arteries are not exposed directly to the marked intra-alveolar pressure changes. Since the bronchial arterioles

can anastomose with the pulmonary arterioles, however, an indirect effect on bronchial arterial flow probably is present,[28,29] although its significance remains to be determined.

Pulmonary arteriovenous fistulas can provide bypaths through which unoxygenated blood from the pulmonary artery may enter the systemic circulation. Such a shunt competes with the normal pulmonary vessels for the right ventricular output.[30] If the resistance to flow through the pulmonary vessels is increased as a result of congenital heart disease, enhanced intrapulmonary air pressure, or because of arteriolar sclerotic changes, flow through the shunt may be enhanced.[31] Redistribution of flow between an artificial fistula and the normal pulmonary vessels has been demonstrated in animals[5] and in isolated lungs.

Gravitational and other obligatory factors are thus seen to provide a variety of patterns which constrain the regulation and distribution of the pulmonary blood flow (Table I). These mechanical factors are probably at the root of many of the clinical syndromes and pathologic changes reflecting the space requirements and the nature of the respiratory membranes of the lungs.

TABLE I. MECHANICAL FACTORS IN FLOW THROUGH A PULMONARY MANIFOLD

CONDITION	FLOW CHARACTERISTICS		REMARKS
	APEX	BASE	
Horizontal (recumbent)	Equalized		
Vertical (upright)	Decreased	Increased	Gravitational factors
Precapillary resistance	Tendency to equalization		Pulmonary arterial pressure must be increased markedly to maintain volume flow
Elevated outlet pressure (pulmonary venous pressure)	Tendency to equalization		Increases congestion of dependent parts Reduces total flow for any given pulmonary arterial pressure
Enhanced intrapulmonary air pressure	Markedly decreased	Markedly increased	Pulmonary vascular resistance increased in irregular fashion

SUMMARY

A specially designed manifold of glass and rubber tubes was perfused with water to simulate certain mechanical aspects of the pulmonary circulation. A pressure head (pulmonary artery) caused flow through four tubes in parallel (pulmonary arterioles) spaced 10 cm. apart.

Vertical positioning of such a pulmonary manifold caused preferential flow through the dependent channels, while little perfused the elevated portions. This effect was exaggerated at low pressure heads. As the pulmonary arterial pressure head was raised, the elevated or apical segments received an improved supply.

Slight to moderate "intrapulmonary" air pressure, uniformly applied to all four "capillaries," shunted flow away from the elevated vessels, and increased that through the dependent tubes. High levels of such air pressure reduced flow as a whole and resulted in a requirement of a high pulmonary arterial pressure if "normal" flow was to be perfused through the "lung."

Elevation of the level of the outlet (pulmonary venous pressure) increased flow through the elevated segments until it equaled that through the base. When the (intrapulmonary) air pressure was then raised, the apical flow was markedly reduced and almost the entire flow shunted through the dependent channels. Certain anomalous patterns of flow due to this combination are described.

Enhancement of precapillary resistance equalized flow through the elevated and dependent portions of the manifold but reduced the total flow for a given pressure head, thereby requiring a higher arterial pressure to maintain perfusion of the system at "normal" flow rates. Elevation of intrapulmonary air pressure in these circumstances had only a limited effect in redistributing flow to the base.

Implications of the effect of mechanical factors on the patterns of flow distribution through a pulmonary system and the effects on pulmonary vascular pressures are outlined. The presumed effects on blood flow in conditions such as pulmonary hypertension subsequent to arteriolosclerosis of the lesser circulation, emphysematoid states, congestive heart failure, orthopnea, the tendencies to pulmonary edema in the dependent portions of the lung, and the predisposition to apical involvement of reinfection tuberculosis are discussed.

SUMMARIO IN INTERLINGUA

Le problema del distribution del fluxo sanguinee in le lecto vascular del pulmones esseva investigate per medio de un serie de "pulmones simulate." Iste studios demonstrava que le configuration del perfusion dependeva significativemente del elevation o abassamento de varie partes del tubage. Illos etiam demonstrava le effectos de elevate pressiones pulmono-arterial, del pression aeree intrapulmonar, del pression pulmono-venose, e del resistentia vascular precapillari. Es describite plure configurationes anormal del fluxo le quales resultava de certe combinationes del mentionate factores. Le datos es discutite in relation con certe aspectos de orthopnea, emphysema, hypertension pulmonar, congestion dependente, disfallimento congestive, e le problema del localisation apical de tuberculosis.

The technical assistance of Bertram Swanson, D.D.S., and Matthew Thompson greatly facilitated the execution of this study. Arnold M. Katz, B.A., assisted in early phases of the experiment.

Discussions with Dr. L. N. Katz were of considerable assistance in the development of the project.

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PHYSICAL BASIS OF BALLISTOCARDIOGRAPHY. II.

The Quantities That Can Be Measured With Different Types of Ballistocardiographs and Their Mutual Relations

H. C. Burger, D.Sc., and A. Noordergraaf, B.Sc.

Utrecht, Netherlands

SINCE it appears from the literature that there might be some confusion in using the terms displacement, velocity, and acceleration (or force) and their relations in ballistocardiographic investigation, a summary of the different possibilities will follow below. It will appear that, if an optimal representation of one of the above-mentioned quantities is desired, then the natural frequency and the damping of the loaded ballistocardiograph (BCG) determine which of these quantities is recorded.

Since we want to know which of the quantities is recorded, we assume for simplicity's sake the binding between body and BCG to be infinitely strong. The subject himself is considered to be a rigid body.

The differential equation of a BCG, loaded with a patient[1] is:

$$M\ddot{x}_c - \beta\dot{x} - Dx = M\ddot{x} \qquad (1)$$

in which x is the displacement, $\dot{x}$ the velocity, and $\ddot{x}$ the acceleration of subject and BCG together with respect to the surroundings (only the longitudinal axis is attended). These quantities are positive in headward direction (to the right in Fig. 1). The quantities x_c, $\dot{x}_c$, and $\ddot{x}_c$, respectively, have the same meaning for the common center of gravity of subject and BCG with respect to the skeleton (positive to the left in Fig. 1). M is the total mass of subject and BCG. $M\ddot{x}_c$ is the force exerted on subject and BCG by the circulation. The frictional force $\beta\dot{x}$ is working in the opposite direction of the velocity $\dot{x}$. Dx is the directive force that drives subject and BCG to the zero position. This force is working in the opposite direction of the displacement x. So, the left side of equation (1) gives the forces working on subject and BCG. They equal the product of the total mass of the moving system M and its acceleration $\ddot{x}$. Equation (1) can also be written in the following form:

$$M\ddot{x} + \beta\dot{x} + Dx = M\ddot{x}_c \qquad (1a)$$

The center of gravity of subject and BCG is assumed to move sinusoidally (term of a Fourier series) with a displacement x_c and an amplitude $|x_c|$. Then x_c can be written in the usual exponential form:

$$x_c = |x_c|\, e^{j\omega t} \qquad (2)$$

$\omega = 2\pi\nu$, ν is the frequency, t the time and j the imaginary unit ($j^2 = -1$).

From the Department of Medical Physics, Physical Laboratory of the University of Utrecht.
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The solution of the differential equation (1a) (valid after a time long enough) is:

$$x = |x| e^{j(\omega t + \varphi)} \tag{3}$$

in which $|x|$ represents the amplitude of this displacement x of subject and BCG. φ is the phase shift (time-lag) between the mass-movement within the subject and the movement of subject and BCG.

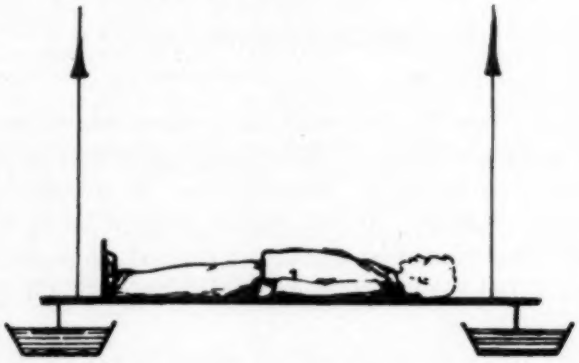


Fig. 1.—Ballistocardiograph loaded with a subject.

The ratio between the amplitude of the displacement of subject and BCG $|x|$ and the amplitude of the displacement of the common center of gravity of subject and BCG $|\dot{x}_c|$ follows from the differential equation (1a) by substituting the formulas (2) and (3). It can be easily proved that:

$$\frac{|x|}{|x_c|} = \left[\left(1 - \frac{\nu_0^2}{\nu^2}\right)^2 + 4\delta^2 \frac{\nu_0^2}{\nu^2}\right]^{-\frac{1}{2}} \tag{4}$$

or

$$\frac{|x|}{|x_c|} = N. \tag{4a}$$

with

$$N = \left[\left(1 - \frac{\nu_0^2}{\nu^2}\right)^2 + 4\delta^2 \frac{\nu_0^2}{\nu^2}\right]^{-\frac{1}{2}}. \tag{5}$$

If subject and BCG are displaced from their zero position and are released without initial velocity, they would oscillate with the natural frequency ν_0 if there were no damping ($\beta = 0$). The frequency with which the common center of gravity moves is represented by ν. The ratio between the damping β and the critical damping β_c is represented by δ.
So

$$\delta = \beta/\beta_c.$$

By differentiating equation (2) with respect to time we find for the velocity of the common center of gravity $\dot{x}_c$:

$$\dot{x}_c = j\omega |x_c| e^{j\omega t}. \tag{6}$$

From this equation the amplitude of the velocity $|\dot{x}_c|$ is found to be:

$$|\dot{x}_c| = \omega|x_c|. \tag{6a}$$

The acceleration $\ddot{x}_c$ of the common center of gravity is found by differentiating $\dot{x}_c$ (equation (6)):

$$\ddot{x}_c = -\omega^2|x_c|e^{j\omega t}. \tag{7}$$

The amplitude of the acceleration is:

$$|\ddot{x}_c| = \omega^2|x_c|. \tag{7a}$$

The internal force that causes the center of gravity to move equals the product of the acceleration of the common center of gravity of subject and BCG and their total mass.

In the same way the velocity $\dot{x}$ of subject and BCG is to be found:

$$\dot{x} = j\omega|x|e^{j\omega t}, \tag{8}$$

with the amplitude for this velocity $|\dot{x}|$:

$$|\dot{x}| = \omega|x|. \tag{8a}$$

For the acceleration is found

$$\ddot{x} = -\omega^2|x|e^{j\omega t}. \tag{9}$$

The amplitude of the acceleration follows from equation (9):

$$|\ddot{x}| = \omega^2|x|. \tag{9a}$$

Equation (4a) gives the ratio $|x|/|x_c|$. With the aid of the equations (6a), (7a), (8a), and (9a) the following ratios can be calculated:

$$\frac{|\dot{x}|}{|x_c|}, \frac{|\ddot{x}|}{|x_c|}, \frac{|x|}{|\dot{x}_c|}, \frac{|\dot{x}|}{|\dot{x}_c|}, \frac{|\ddot{x}|}{|\dot{x}_c|}, \frac{|x|}{|\ddot{x}_c|}, \frac{|\dot{x}|}{|\ddot{x}_c|} \quad \text{and} \quad \frac{|\ddot{x}|}{|\ddot{x}_c|}.$$

We find:

$$|x_c| : |\dot{x}_c| : |\ddot{x}_c| : |x| : |\dot{x}| : |\ddot{x}| = 1 : \omega : \omega^2 : N : N\omega : N\omega^2 \tag{10}$$

in which N is dependent on the frequency (formula (5)).

There will be a time-lag between the phenomena occurring within the body and the movement of body and BCG. This time-lag can be indicated by an angle φ, the phase shift. The phase shift φ between the displacement of the common center of gravity of subject and BCG (x_c) and the displacement of subject and BCG (x) is to be calculated by substituting the formulas (2) and (3) in the differential equation (1a). It follows:

$$\operatorname{tg} \varphi = \frac{2\delta\nu_0\nu}{\nu^2 - \nu_0^2} \tag{11}$$

The phase shift φ^* in all other cases can be read from Fig. 2. The figure is rotating in the direction of the arrow. φ^* is positive in the direction of the arrow and is counted from the quantity with index c to the quantity without index. For instance: the phase shift φ^* between the acceleration of the common center of gravity ($\ddot{x}_c$) and the displacement of subject and BCG (x) equals:

$$\varphi^* = \varphi + 180°$$

Three main types of ballistocardiographs are used:

A. The *low-frequency*, critically or less than critically damped BCG.[1] The natural frequency ν_o of the loaded BCG is low with respect to the frequency of the heart ($\nu_o \ll 1$c/s, $\delta \leqslant 1$).

B. The *middle-frequency* critically damped BCG according to Nickerson. The natural frequency of the loaded BCG is neither high nor low with respect to the frequency of the heart ($\nu_o = 1$–2 c/s, $\delta = 1$).

C. The *high-frequency* BCG. The natural frequency of the loaded BCG is high with respect to the frequency of the heart. According to Starr ν_o is about 15 c/s. Dock has chosen a rigid underlayer as BCG. So, in the extreme case $\nu_o = \infty$; the BCG does not move. Therefore Dock must record the movement of the subject. If the binding between body and BCG were infinitely strong, as has been assumed earlier in this paper, then the subject would not move when a BCG according to Dock is used. So, this BCG does not fit in the scheme of this paper. It will be discussed in a following one.[2]

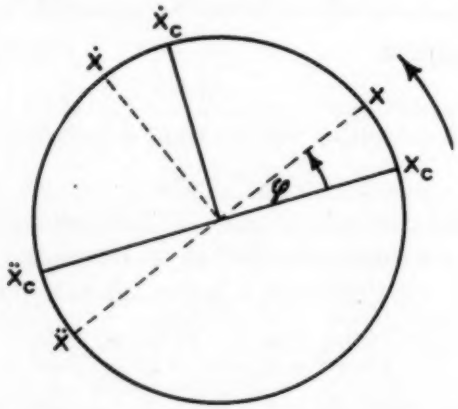


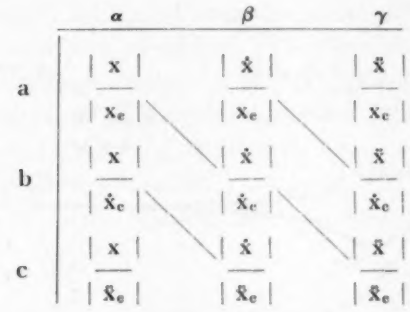
Fig. 2.—If the displacement (x), the velocity ($\dot{x}$), or the acceleration ($\ddot{x}$) of subject and BCG is recorded, the phase shift between the recorded quantity and each of the quantities displacement (x_c), velocity ($\dot{x}_c$), and acceleration ($\ddot{x}_c$) of the common center of gravity of subject and BCG equals the angle φ*. This angle can be found by measuring the angle between the quantity of the common center of gravity one is interested in, and the quantity one records. The angle must be measured in the direction of the arrow.

In this paper we will consider of the high-frequency BCG's only the less than critically damped, high-frequency BCG according to Starr.

We will calculate how each of the three types of BCG's represents:

(a) the displacement of the common center of gravity (x_c);
(b) the velocity of the common center of gravity ($\dot{x}_c$);
(c) the acceleration of the common center of gravity ($\ddot{x}_c$), if either the displacement (x) of subject and BCG or its velocity ($\dot{x}$) or its acceleration ($\ddot{x}$) is recorded. Mostly one of the three last-mentioned quantities are consciously or unconsiously recorded.

So there are the following nine amplitude characteristics for each type of BCG (the rows are indicated with a,b,c, the columns with α, β, γ):



Moreover, there are nine corresponding phase characteristics for each type of BCG. The amplitude and phase characteristics $a\alpha$, $b\beta$, and $c\gamma$ are equal. Likewise $a\beta$ and $b\gamma$. Also $b\alpha$ and $c\beta$. The equal ones are joined by broken lines. These equalities follow from formula (10).

The remaining five different amplitude and phase characteristics are represented:

1. In Figs. 3, 4, 5, 6, and 7 for a *low-frequency* BCG with a natural frequency of 0.3 c/s. The damping is critical ($\delta = 1.0$). In Fig. 3 also the characteristics for a less than critical damping ($\delta = 0.4$) are represented. (The ordinates of the amplitude characteristics are calculated in the c.g.s. system, so the amplitude in Fig. 5, e.g., is given in sec^2).

2. In Figs. 8, 9, 10, 11, and 12 for a *middle-frequency* BCG. The natural frequency of the loaded BCG is 1.5 c/s. Two values are chosen for the damping: (1) critical damping ($\delta = 1$) according to Nickerson; (2) damping far more than critical ($\delta = 5$). For the reason of this choice see below.

3. In Figs. 13, 14, 15, 16, and 17 for a *high-frequency* less than critically damped BCG ($\delta = 14.10^{-3}$). The natural frequency of the loaded BCG is 15 c/s. (The properties represented in these figures can also be represented by other methods.)

An amplitude characteristic has to meet the requirement that it must be flat, for instance within 20 per cent, in the frequency range one is interested in (from about 1 c/s to about 20 c/s). The phase shift must be smaller than, e.g., 20 degrees in the same frequency range. (If the phase shift is not diverging more than 20 degrees from 180 degrees, the curve is correct, for a phase shift of 180 degrees means that the curve is inverted.)

It will now be ascertained systematically which frequency characteristics meet these requirements.

A. The *low-frequency* BCG. From the characteristics in Figs. 3, 4, 5, 6, and 7 it follows that only the characteristics of Fig. 3 can be used. Moreover, it follows from Fig. 3 that the characteristics become better if we choose the damping somewhat smaller than critical. If $\delta = 0.4$ is chosen, then the characteristics are quite satisfactory.

B. The *middle-frequency* BCG. If the value of δ is chosen so that the damping is critical, as is done by various investigators, none of the frequency characteristics meet the conditions (Figs. 8, 9, 10, 11, and 12). Only if a very heavy damping is used ($\delta \geqslant 5$) the frequency characteristics in Fig. 9 are useful, after proper calibration of the apparatus.

Fig. 3.

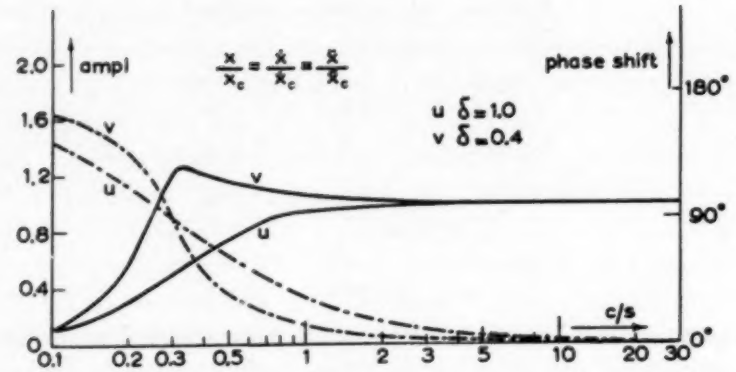


Fig. 4.

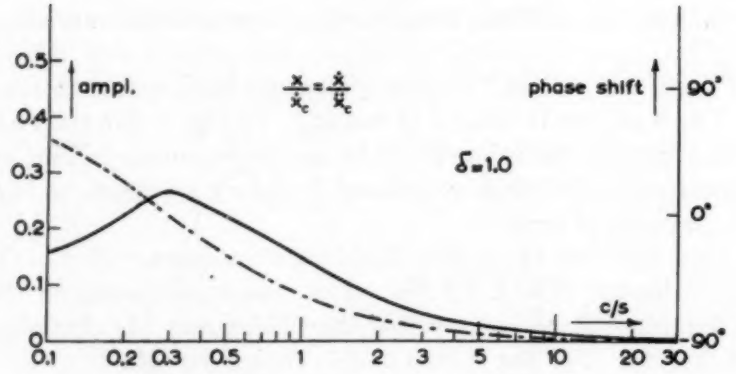


Fig. 5.

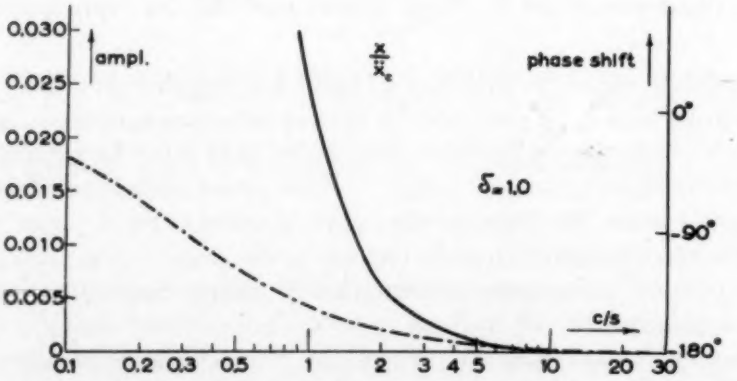


Fig. 3.—Amplitude characteristics (solid lines) and phase characteristics (broken lines) of a *low-frequency* BCG (ν_0 = 0.3 c/s; δ = 1.0 and 0.4), indicating how the displacement of the center of gravity, its velocity, and its acceleration are represented by the displacement, the velocity, and the acceleration of the BCG, respectively.

Fig. 4.—Amplitude characteristic (solid line) and phase characteristic (broken line) of a *low-frequency* BCG (ν_0 = 0.3 c/s; δ = 1.0), indicating how the velocity of the center of gravity and its acceleration are represented by the displacement and the velocity of the BCG, respectively.

Fig. 5.—Amplitude characteristic (solid line) and phase characteristic (broken line) of a *low-frequency* BCG (ν_0 = 0.3 c/s; δ = 1.0), indicating how the acceleration of the center of gravity is represented by the displacement of the BCG.

C. The *high-frequency* BCG. From the characteristics in Figs. 13, 14, 15, 16, and 17 it follows that only those of Fig. 15 are suitable if the natural frequency of the loaded BCG (ν_o) is high enough and the apparatus is calibrated properly. So, if a reliable representation of the phenomena is wished for frequencies up to 20 c/s the natural frequency must be at least about 25 c/s. In the case of Fig. 15 ($\nu_o = 15$ c/s) the representation is reliable to about 10 c/s. Therefore, if a correct measurement of the quantities is required, the following possibilities remain:

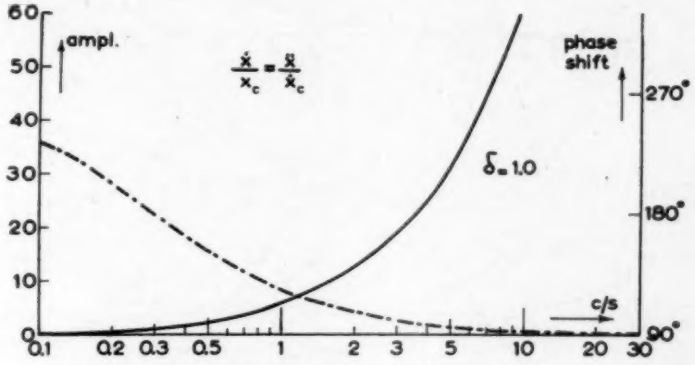


Fig. 6.

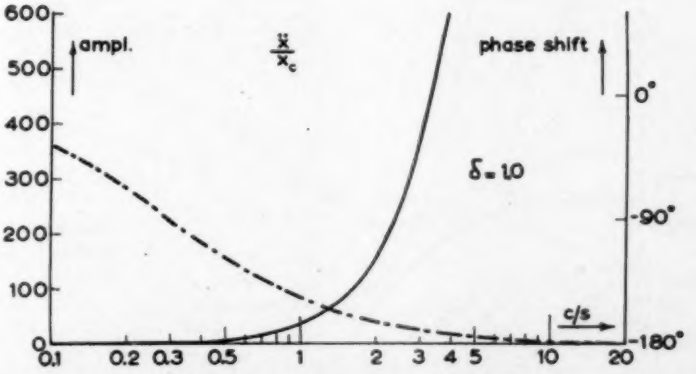


Fig. 7.

Fig. 6.—Amplitude characteristic (solid line) and phase characteristic (broken line) of a *low-frequency* BCG ($\nu_o = 0.3$ c/s; $\delta = 1.0$), indicating how the displacement of the center of gravity and its velocity are represented by the velocity and the acceleration of the BCG, respectively.

Fig. 7.—Amplitude characteristic (solid line) and phase characteristic (broken line) of a *low-frequency* BCG ($\nu_o = 0.3$ c/s; $\delta = 1.0$), indicating how the displacement of the center of gravity is represented by the acceleration of the BCG.

A. The *low-frequency* BCG (natural frequency about 0.3 c/s, δ about 0.4). (See Fig. 3.)

a. If the displacement of subject or BCG (x) is recorded, the obtained curve represents the displacement of the common center of gravity of subject and BCG (x_c), caused by the heart action.

b. If the velocity of subject or BCG ($\dot{x}$) is recorded, the obtained curve represents the velocity of the common center of gravity ($\dot{x}_c$).

Fig. 8.

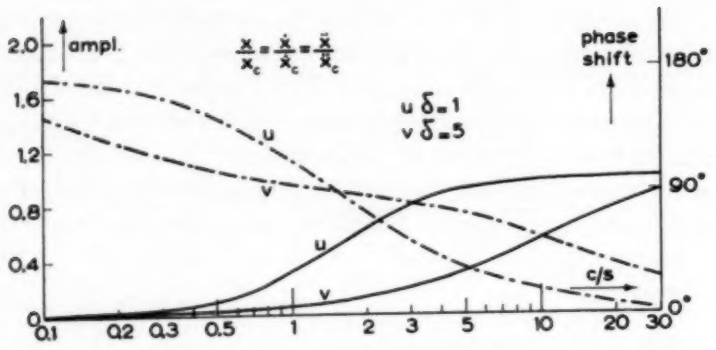


Fig. 9.

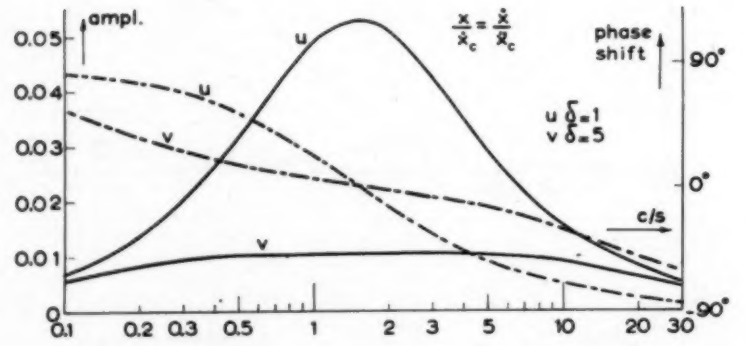


Fig. 10.

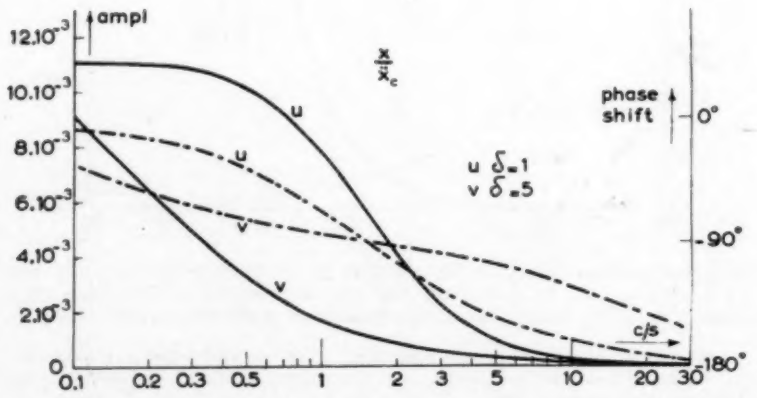


Fig. 8.—Amplitude characteristics (solid lines) and phase characteristics (broken lines) of a *middle-frequency* BCG (ν_0 = 1.5 c/s; δ = 1 and 5), indicating how the displacement of the center of gravity, its velocity, and its acceleration are represented by the displacement, the velocity, and the acceleration of the BCG, respectively.

Fig. 9.—Amplitude characteristics (solid lines) and phase characteristics (broken lines) of a *middle-frequency* BCG (ν_0 = 1.5 c/s; δ = 1 and 5), indicating how the velocity of the center of gravity and its acceleration are represented by the displacement and the velocity of the BCG, respectively.

Fig. 10.—Amplitude characteristics (solid lines) and phase characteristics (broken lines) of a *middle-frequency* BCG (ν_0 = 1.5 c/s; δ = 1 and 5), indicating how the acceleration of the center of gravity is represented by the displacement of the BCG.

c. If the acceleration of subject or BCG ($\ddot{x}$) is recorded, the obtained curve represents the acceleration of the common center of gravity ($\ddot{x}_c$).

(In principal, it is of no importance in which way x, $\dot{x}$, or $\ddot{x}$ is recorded. It is only a matter of experimental methods, and will not be discussed here.)

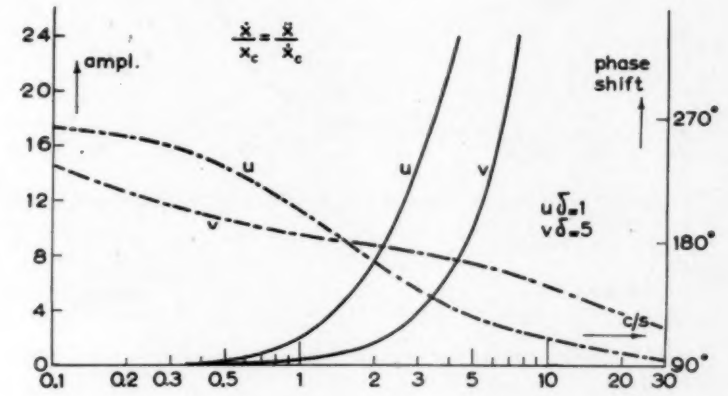


Fig. 11.

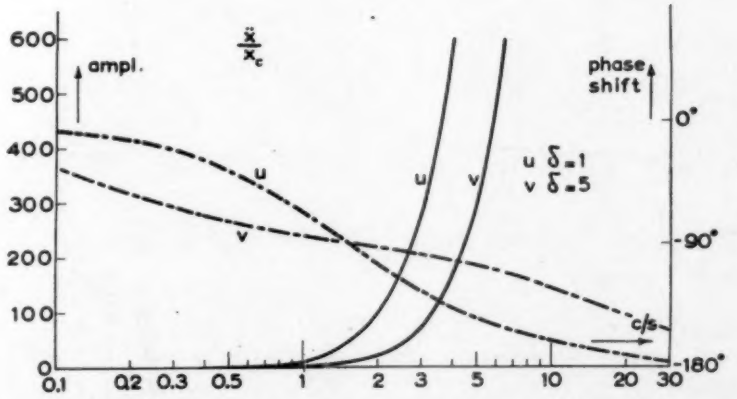


Fig. 12.

Fig. 11.—Amplitude characteristics (solid lines) and phase characteristics (broken lines) of a *middle-frequency* BCG (ν_o = 1.5 c/s; δ = 1 and 5), indicating how the displacement of the center of gravity and its velocity are represented by the velocity and the acceleration of the BCG, respectively.

Fig. 12.—Amplitude characteristics (solid lines) and phase characteristics (broken lines) of a *middle-frequency* BCG (ν_o = 1.5 c/s; δ = 1 and 5), indicating how the displacement of the center of gravity is represented by the acceleration of the BCG.

The curves mentioned under a, b, and c are mathematically related: The curve under b can be obtained by differentiating once the curve under a. The curve under c can be obtained by differentiating once the curve under b or, of course, by differentiating twice the curve under a.

The reverse, the curve under b and a can be obtained from the curve under c by integrating once and twice, respectively. This is schematically represented here.

Fig. 13.

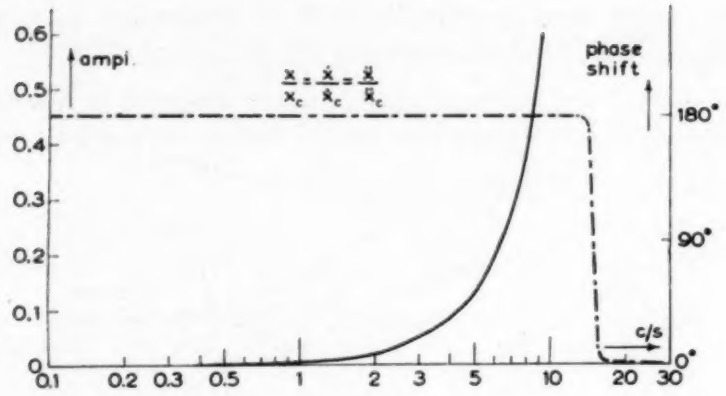


Fig. 14.

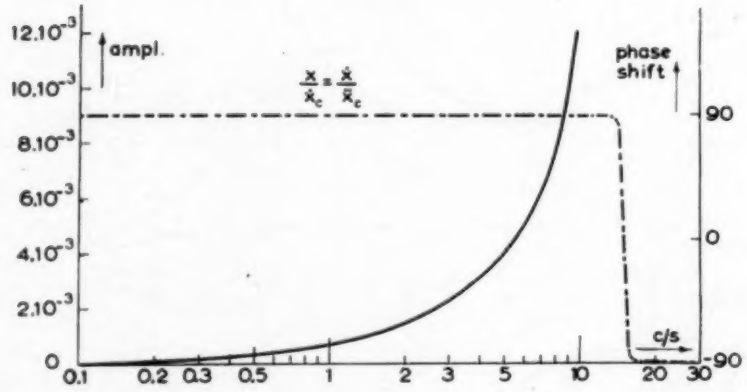


Fig. 15.

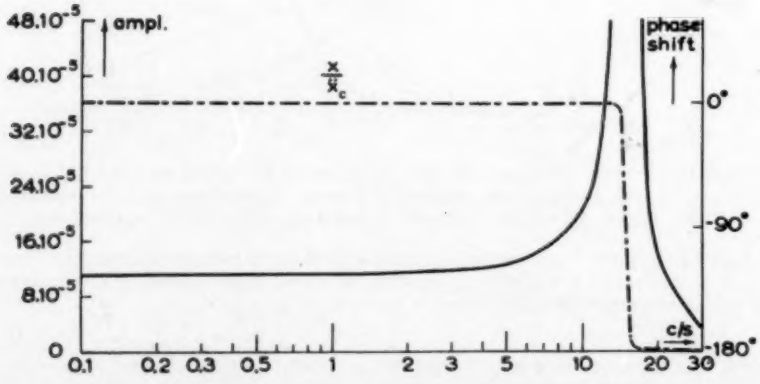
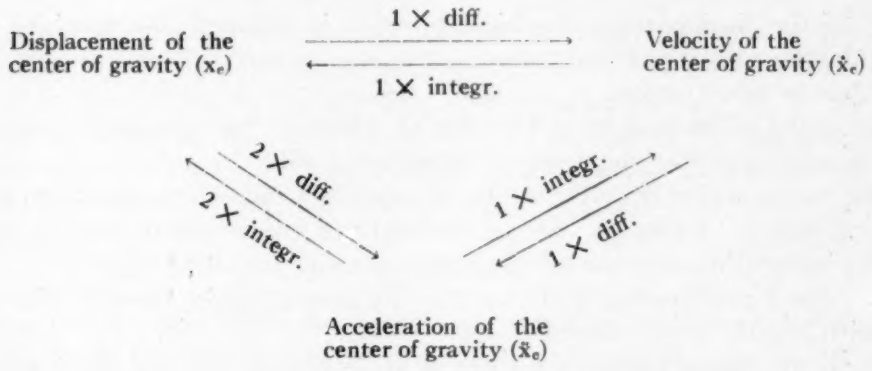


Fig. 13.—Amplitude characteristic (solid line) and phase characteristic (broken line) of a *high-frequency* BCG (ν_0 = 15 c/s; $\delta = 14.10^{-3}$), indicating how the displacement of the center of gravity, its velocity, and its acceleration are represented by the displacement, the velocity, and the acceleration of the BCG, respectively.

Fig. 14.—Amplitude characteristic (solid line) and phase characteristic (broken line) of a *high-frequency* BCG (ν_0 = 15 c/s; $\delta = 14.10^{-3}$), indicating how the velocity of the center of gravity and its acceleration are represented by the displacement and the velocity of the BCG, respectively.

Fig. 15.—Amplitude characteristic (solid line) and phase characteristic (broken line) of a *high-frequency* BCG (ν_0 = 15 c/s; $\delta = 14.10^{-3}$), indicating how the acceleration of the center of gravity is represented by the displacement of the BCG.



B. The *middle-frequency* BCG (natural frequency about 1.5 c/s, δ greater than 5). (See Fig. 9.)

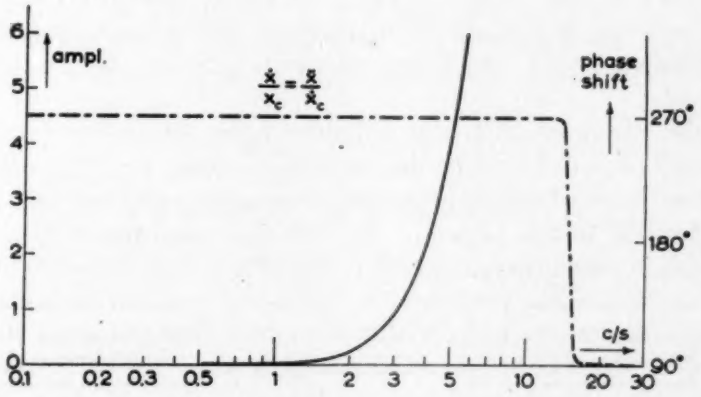


Fig. 16.

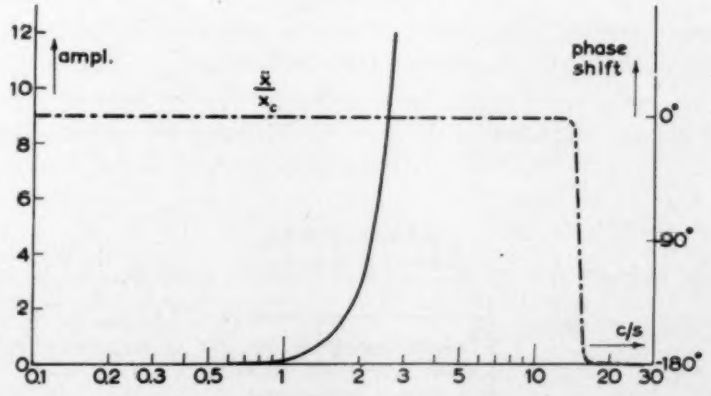


Fig. 17.

Fig. 16.—Amplitude characteristic (solid line) and phase characteristic (broken line) of a *high-frequency* BCG (ν_0 = 15 c/s; $\delta = 14.10^{-2}$), indicating how the displacement of the center of gravity and its velocity are represented by the velocity and the acceleration of the BCG, respectively.

Fig. 17.—Amplitude characteristic (solid line) and phase characteristic (broken line) of a *high-frequency* BCG (ν_0 = 15 c/s; $\delta = 14.10^{-3}$), indicating how the displacement of the center of gravity is represented by the acceleration of the BCG.

b. If the displacement of subject or BCG is recorded, the obtained curve represents the velocity of the common center of gravity of subject and BCG, caused by the heart action.

c. If the velocity of subject or BCG is recorded, the obtained curve represents the acceleration of the common center of gravity.

The curves under b and c are mathematically related in the same way as described above. Moreover, the displacement of the center of gravity can be found by integrating once the curve under b or twice the curve under c.

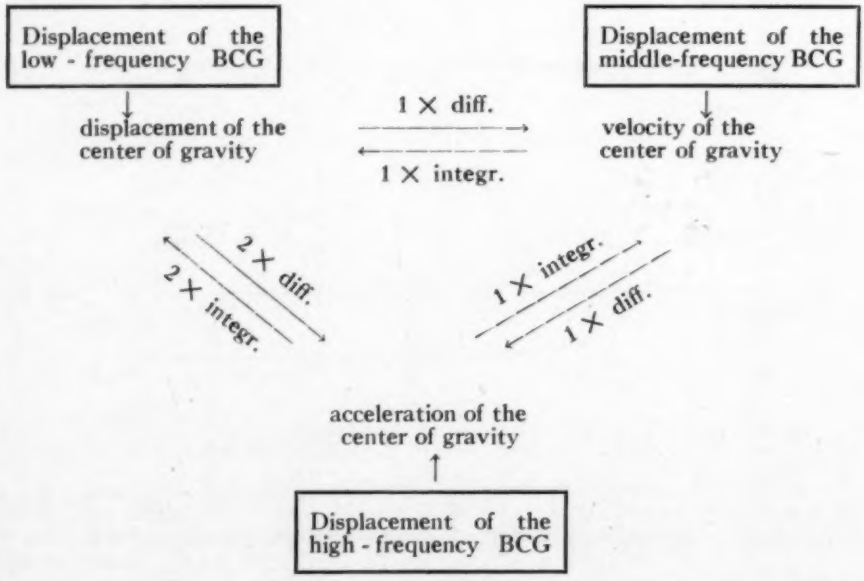
C. The *high-frequency* BCG (natural frequency higher than 20 c/s, δ very small with respect to 1.) (See Fig. 15.)

c. If the displacement of subject or BCG is recorded, the obtained curve represents the acceleration of the common center of gravity of subject and BCG.

The velocity and the displacement of the center of gravity can be obtained from this curve by integrating it once and twice respectively.

From the above it follows that the displacement, the velocity, and the acceleration of the common center of gravity of subject and BCG can be found by means of a low-frequency, a middle-frequency, and a high-frequency BCG, if the natural frequency and the degree of damping meet the requirements mentioned previously.

The results obtained with the different types of ballistocardiographs are mutually related provided that subject and BCG move exactly in the same way, and the different parts of the body do not move with respect to each other (as has been assumed earlier in this paper). The relation when the *displacement* of each type is recorded is shown here.



Analogous schemes can be made if other quantities are recorded (27 schemes in all).

In fact, it is not realizable that the movement of the subject and of the BCG are exactly the same. The stronger the binding of the BCG to the surroundings

(the higher the natural frequency and the heavier the damping), the greater the difference in movement (the "relative movement"). As a result of this relative movement the amplitude characteristic of the high-frequency BCG is not half as excellent as in Fig. 15. Because of this phenomenon it is preferable to use a low-frequency BCG if a correct representation of the occurrences is desired. This point will be worked out in a following paper.[2]

SUMMARY

It is investigated which quantity concerning the common center of gravity of a subject is recorded by a low-frequency, a middle-frequency, and a high-frequency ballistocardiograph. It appeared that the displacement of these types of ballistocardiographs represents the displacement, the velocity, and the acceleration of the center of gravity, respectively, if the external circumstances meet the given requirements. Corresponding amplitude and phase characteristics are calculated.

A relation between the quantities of displacement, velocity, and acceleration of the center of gravity is deduced, from which follows the method by which these three quantities can be found from each of the three types of ballistocardiographs.

In this paper the binding between body and ballistocardiograph is assumed to be infinitely strong.

SUMMARIO IN INTERLINGUA

Esseva investigate qual quantitate relative al commun centro de gravitate del subjecto es registrate per le ballistocardiographo a basse, a median, e a alte frequentia. Il pareva que le displaciamento de iste tres typos de cardiographo representa, respectivemente, le displaciamento, le velocitate, e le acceleration del centro de gravitate, providite que le conditiones externe satisface le correspondente requirimentos. Le correspondente amplitude e characteristicas phasic esseva calculate.

Esseva deducite un relation inter displaciamento, velocitate, e acceleration del centro de gravitate. Per medio de iste relation le tres mentionate quantitates es determinabile con omne le tres typos de ballistocardiographo.

In le deductiones del presente reporto il es assumite que le fortia ligante inter corporee balli stocardiographo es infinite.

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1. Burger, H. C., Noordergraaf, A., and Verhagen, A. M. W.: Physical Basis of the Low-Frequency Ballistocardiograph, AM. HEART J. 46:71, 1953.
2. Burger, H. C., Noordergraaf, A., Korsten, J. J. M., and Ullersma, P.: Physical Basis of Ballistocardiography. IV. In preparation.

Clinical Reports

BIDIRECTIONAL TACHYCARDIA

EMANUEL HELLMAN, M.D., F.A.C.P., AND ARTHUR LIND, M.D.

NEW YORK, N. Y.

BIDIRECTIONAL ventricular tachycardia, or paroxysmal ventricular tachycardia with rhythmic alternation in the direction of the ventricular complexes, is one of the least common of all arrhythmias. There have been thirty-four such cases reported in the literature.[1,2]

The appearance of this arrhythmia has been associated with severe myocardial damage in almost every case. In twenty-six of the thirty-four cases previously reported death occurred soon after the arrhythmia was discovered. In four others, death occurred within two to twenty-seven months. Antecedent atrial fibrillation was observed in sixteen of the reported cases. In twenty-eight of the thirty-four reported cases, digitalis was being administered at the time the arrhythmia was discovered. Although some of these patients received digitalis in toxic doses, in the majority the drug was administered within the average therapeutic range, and in a few the arrhythmia occurred in patients who had not received digitalis. These tachycardias, as well as extrasystoles, were not observed in reported cases of suicide by digitalis,[3] and they cannot be produced in the normal animal unless the myocardium has been previously injured.[4] Thus the quantity of digitalis is not the factor of exclusive importance. It is the status of the heart muscle that is chiefly responsible for the development of this arrhythmia.

This case history is presented for the purpose of showing the response of bidirectional ventricular tachycardia to carotid sinus pressure, intravenous magnesium sulfate, and potassium chloride infusion, and to discuss the mechanism of this arrhythmia.

CASE REPORT

M. Z., a 68-year-old white male, entered the Gouverneur Hospital for the first time on Nov. 6, 1952, in moderately severe congestive failure which responded slowly to Digoxin, salt-free diet, and mercurials. He was discharged on Dec. 4, 1952, with a diagnosis of hypertensive arteriosclerotic heart disease. At that time he was fibrillating slowly with a ventricular rate of eighty and no signs of digitalis toxicity.

From the Medical Service and Cardiac Clinic, Gouverneur Hospital, New York, N. Y.
Received for publication May 6, 1955.

He was readmitted on Jan. 15, 1954, because of increasing dyspnea, cough, and edema. Physical examination revealed an elderly male who appeared acutely ill; severely dyspneic, orthopneic, and cyanotic; lethargic and mentally confused. Temperature was 102.8° F., and blood pressure 165/110 mm. Hg. The neck veins were distended and filled from below, and there was a positive hepatojugular reflux. There were dullness and decreased breath sounds over the left lower lobe posteriorly with wheezing and rhonchi throughout both lung fields. The heart was massively enlarged with the point of maximal impulse in the sixth intercostal space at the anterior axillary line. There were no thrills or murmurs. The rhythm was grossly irregular with a ventricular rate of 160 and a pulse rate of 100. The liver extended 5 cm. below the costal margin, and there was 4-plus leg edema. Both legs showed extensive cellulitis with multiple superficial ulcerations. Neither dorsalis pedis or posterior tibial artery could be felt.

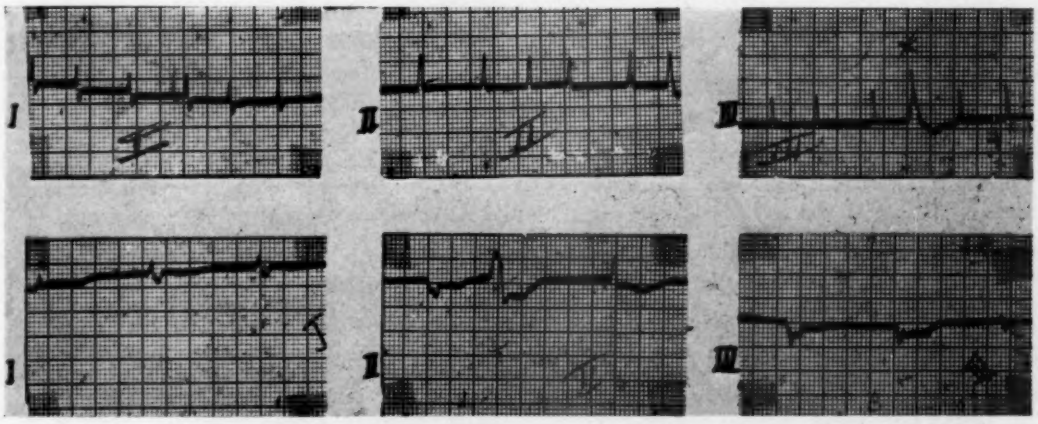

Fig. 1.—*A*, Jan. 15. Initial tracing. Atrial fibrillation; ventricular rate 160 per minute; occasional ventricular premature contraction. *B*, Feb. 8. Multifocal ventricular premature contractions following digitalization.

Laboratory findings: Urinalysis showed a specific gravity of 1,010 and a 2-plus albumin. Hemoglobin, 14.2 grams; white blood cells 25,700 with 91 per cent polynuclear cells; sedimentation rate 38 mm./hour; hematocrit 45 per cent. The Bromsulphalein test showed 16 per cent retention in 45 minutes. Carbon dioxide combining power was 115 and 70 volumes per cent. All other blood chemistries were normal. Six blood cultures were negative. The venous pressure was 220 mm. of water, and the circulation time was 23 seconds (Decholin).

An electrocardiogram (Fig. 1) revealed atrial fibrillation with a ventricular rate of 160.

The infection was treated with large doses of antibiotics. The heart failure was treated with diet, oxygen, mercurials, and digitalization with intravenous Cedilanid (0.8 mg.) followed by oral digitoxin, (0.2 mg. daily). On Jan. 26, an electrocardiogram showed atrial fibrillation with a ventricular rate of 60, and S-T and T changes attributable to digitalis. On Feb. 8, the electrocardiogram showed multifocal ventricular extrasystoles (Fig. 1). On Feb. 10, the patient had an episode of paroxysmal tachycardia with a rate of 160 which disappeared with carotid sinus pressure. This recurred on Feb. 11, when the electrocardiogram revealed bidirectional ventricular tachycardia (Fig. 2,*A*). Carotid sinus pressure was applied with abrupt cessation of the paroxysm as shown in Fig. 2,*B*. After fifteen minutes the bidirectional ventricular tachycardia returned (Fig. 2,*E*) and at this time 20 c.c. of 20 per cent magnesium sulfate was rapidly injected intravenously, resulting in abrupt cessation of the paroxysm (Fig. 2,*F*). Twenty minutes after administration of magnesium sulfate, an infusion containing 40 meq. of potassium chloride in 1,000 c.c. of 5 per cent glucose in water was begun and was administered over a twelve-hour period. At this point, digitalis having been stopped when the tachycardia was recognized, the infusion was discontinued and oral administration of potassium chloride, one gram three times

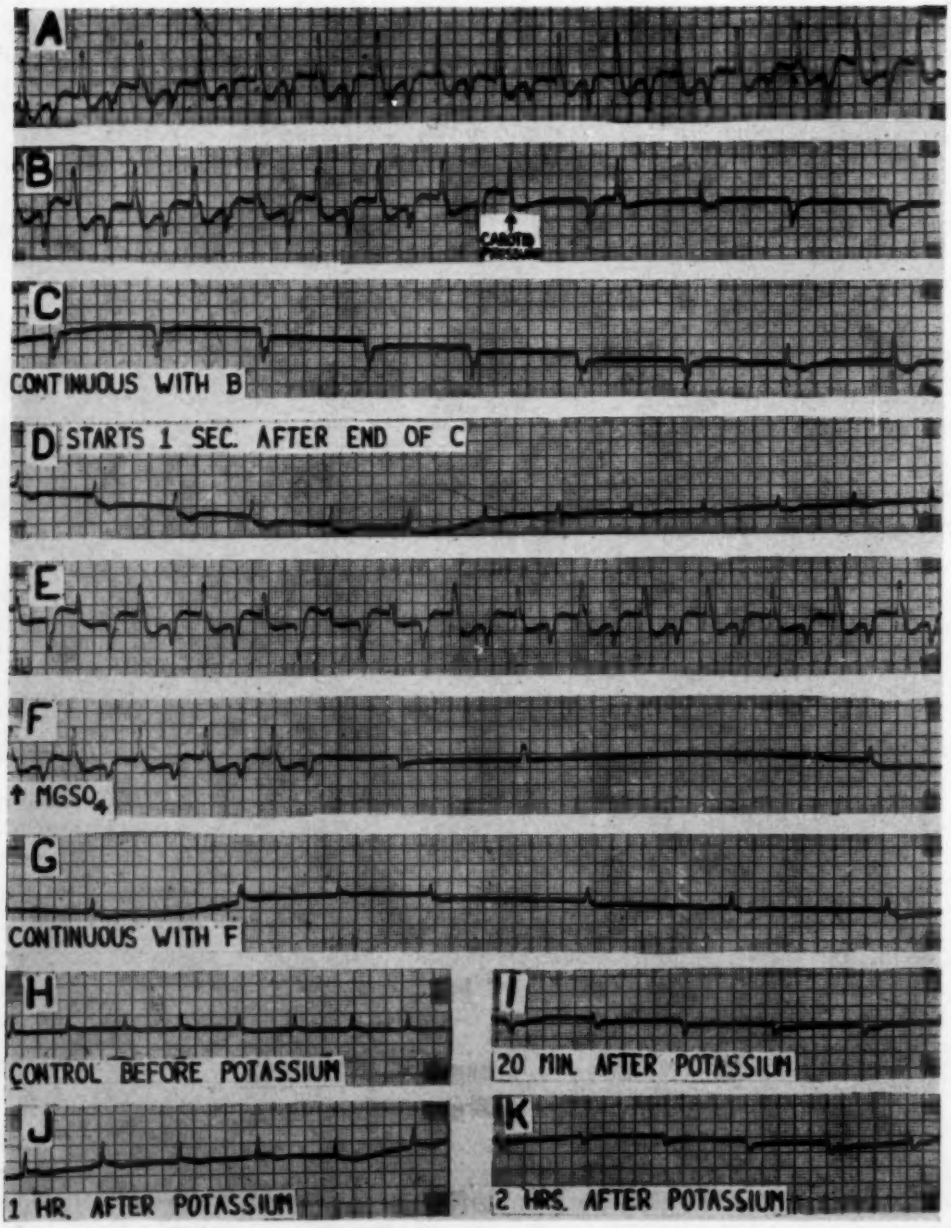

Fig. 2.—(All strips are Lead II). *A*, Bidirectional tachycardia. Over-all rate 158 per minute. *B*, Fixed relationship between complexes as described in text. Arrow indicates application of carotid sinus pressure. *C*, Upward directed complexes abolished. Downward-directed complexes remain for a short time but at much slower rate (45 per minute). *D*, This is followed by upward directed complexes, supraventricular in form with slightly irregular rhythm suggesting either nodal focus or reversion to atrial fibrillation. *E*, The bidirectional tachycardia then returns. *F*, Intravenous magnesium sulfate is shortly followed by a few escape beats, a long period of asystole, followed by irregularly spaced beats of supraventricular form (*G*); and then by regular supraventricular rhythm with a rate of 83 (*H*). *I*, twenty minutes after beginning of potassium infusion there is a change in rate, shape, and direction of complexes. *J*, This rhythm, except for a brief period of reversion to atrial fibrillation, is maintained throughout the period of potassium infusion.

a day was initiated. By Feb. 15, the patient was again in severe heart failure, and digitalis was reinstituted in the form of Digoxin, 0.5 mg. daily. This was continued without further evidence of toxicity.

In the meantime, the ulcerations on the right leg had progressed and become gangrenous, necessitating above-the-knee amputation on March 1. On March 5, he developed pneumonia with rising temperature, dyspnea, and cyanosis and expired on March 8. Permission for autopsy was denied.

DISCUSSION

Analysis of the electrocardiogram at the height of the paroxysm (Fig. 2,*A*) shows a perfectly regular alternation of ventricular complexes with an over-all rate of 158 per minute. The rate of the upward-directed complexes is exactly the same as the rate of the downward-directed complexes (79 per minute). The interval between the upward-directed complexes is always 0.76 sec., and this is exactly equal to the interval between the downward-directed complexes. The interval between an upward-directed complex and the succeeding downward-directed complex is stable at 0.36 sec. The interval between a downward-directed complex and the following upward-directed complex is also stable but is slightly longer, measuring 0.4 sec. The duration of QRS in the downward-directed complexes measures 0.12 sec. In the upward-directed complexes the onset of the T wave is not distinct, but if this is taken at the point where the S wave becomes abruptly thickened the duration of QRS also measures 0.12 sec.

Of the many theories that have been advanced to explain the phenomenon of the alternating complexes in bidirectional ventricular tachycardia, the one most generally accepted until recently has been that there are two foci of impulse formation functioning alternately, one in the right and one in the left ventricle.[5,6]

Recently this concept has been challenged, particularly following observed effects of carotid sinus pressure upon the arrhythmia.[1,2] In this case, pressure upon the carotid sinus abruptly ended the paroxysm (Fig. 2,*B*). This form of vagal stimulation was applied in two of the previously reported cases. In one of these, the upward-directed complexes were abolished while the downward-directed complexes remained.[1] In the second, there was an abrupt return of the original atrial fibrillation, followed by nodal rhythm with upward-directed complexes, alternating with runs of regularly spaced downward-directed complexes resembling those of the paroxysm.[2] From these observations it was concluded that there were two foci functioning: one, which was abolished by carotid pressure in the supraventricular atrioventricular nodal tissue, and the other in the ventricles. It was, therefore, proposed that the term paroxysmal tachycardia with rhythmic alternation in the direction of the ventricular complexes be used instead of bidirectional ventricular tachycardia.[1,2]

The third most likely explanation for the mechanism responsible for the alternating ventricular complexes is that there is only one center of impulse formation situated above the bifurcation of the bundle of His with alternate conduction down the right and left bundle branches.[5,7] From the analysis of the electrocardiogram in this case the following points may be cited in favor of this theory.

1. The upward-directed complexes observed during the paroxysm (Fig. 2,*A* and *E*) differ completely in form from the upward-directed ventricular extrasystoles that were seen prior to the onset of the arrhythmia (Fig. 1). On the other hand, the downward-directed complexes observed during the paroxysm resemble, to a great extent, the downward-directed ventricular premature contractions seen prior to the onset of the arrhythmia. In some of the reported cases distinct similarity in form and coupling time of the ventricular extrasystoles observed prior to the onset of the arrhythmia and the complexes of the paroxysm were noted as evidence of a ventricular origin of the focus.[6] The difference in form noted in this case indicates, at least in the case of the upward-directed complexes, an origin above the bifurcation of the bundle of His.

2. The regular alternation of two kinds of ventricular complexes, it has been stated, is difficult to explain on the basis of impaired conduction alone.[6] However, the existence of two foci functioning alternately at exactly the same rate is even more unlikely.

3. Prolonged intraventricular conduction (QRS measures 0.12 sec.) suggests an origin below the bifurcation, but this is just as characteristic of abnormal conduction, as in bundle branch block.

4. The interval between an upward-directed complex and the succeeding downward-directed complex is always 0.36 sec., whereas the interval between a downward-directed complex and the succeeding upward-directed complex is always 0.4 sec. This alternation in the length of ventricular diastole mitigates strongly the concept of a single focus. In fact, on the basis of this finding a double circus movement in the ventricles has been suggested as the mechanism.[8] However, in some of the reported cases the rhythm has been perfectly regular. Such exact rhythmicity would be difficult to explain except on the basis of a single focus. Furthermore, even when the rhythm of the ventricular diastole alternates, an interchanging action of two centers cannot be assumed, since it has been shown that activity of one center (in the auricle) may be followed by alternation in the form of the ventricular complexes both in regard to shape and rhythm.[3] In addition, a form of rhythmic alternating tachycardia has been described in which both types of ventricular complexes go in the same direction.[3]

In view of the fixed coupling and the presence of an intraventricular conduction defect, the possibility of a re-entry phenomena to explain the mechanism of the arrhythmia might also be entertained. Thus the dominant rhythm would be supraventricular with each beat followed by a re-entry beat, as in ventricular bigeminy.[9-12]

5. Carotid sinus pressure abolished the upward-directed complexes while the downward-directed complexes still remained for a short time (Fig. 2,*C*). However, the rate of the downward-directed complexes was considerably slowed, indicating that these were also affected by the vagal stimulation and suggesting a possible origin above the bifurcation. This is followed by complexes which appear supraventricular in form, with a very slight irregularity in rhythm, suggesting either a nodal focus or a reversion to atrial fibrillation.

In addition to the two cases mentioned in which carotid sinus pressure was exerted, Bellet[13] cites two others in which the paroxysm was completely stopped.

It is well known that vagal stimulation will stop supraventricular tachycardia, but it is thought that such stimulation has no effect upon ventricular tachycardia. In mammals, if the A-V bundle is severed, vagal stimulation has no effect upon the ventricular rate.[14] Also, observations in complete heart block indicate that in the labile type of idioventricular focus, subject to vagal influences, the shape of the ventricular complexes suggests an origin above the bifurcation of the bundle of His; whereas in the stable type, not affected by alternations in vagal tone, the form of the complexes suggests an origin below the bifurcation.[15-17]

Although these observations do not preclude the possibility of two foci, one above and one below the bifurcation of the bundle of His, they do suggest that the entire mechanism may be located in the bundle above the bifurcation. While it has been suggested that there may be two independent foci located at the bifurcation with alternate conduction over each bundle branch in successive cycles,[5,18] it seems more likely that only one such focus exists and that the impulse is conducted alternately down the right and left bundle branches.

In all of the cases cited in which carotid sinus pressure was applied, the arrhythmia was partially or completely aborted. It is suggested, therefore, that in future cases where such pressure effectively aborts the paroxysm, treatment with cholinergic drugs may prevent recurrence of the arrhythmia.

Most of the reported cases of bidirectional ventricular tachycardia have been associated with digitalis. It is known that digitalis causes ventricular ectopic beats as an early manifestation of toxicity and that this is often followed by regular coupled rhythm if the administration of the drug is continued, particularly if atrial fibrillation be present. It is also known that digitalis may act in these patients to depress the conducting system. This depression is enhanced by the ventricular extrasystoles, which decrease ventricular output, thereby increasing anoxia.[16] Such action, in patients with already damaged tissues, may produce a high degree of A-V block with the result that a pacemaker in the bundle suddenly takes over. In view of these observations it is proposed that the simplified term bidirectional tachycardia be used to describe this arrhythmia.

EFFECTS OF MAGNESIUM

Rapid intravenous injection of 20 c.c. of 20 per cent magnesium sulfate caused almost immediate cessation of the paroxysm. After a few escape beats there ensued a long period of asystole following which a slow irregular rhythm was initiated with complexes which are supraventricular in form and upright in direction (Fig. 2,*F* and *G*). The rate then rapidly increased, and the rhythm became perfectly regular at a rate of 83 per minute, apparently a rapid nodal rhythm (Fig. 2,*H*).

Experimentally, it has been demonstrated that magnesium salts in large doses depress conduction and depress or abolish abnormal impulse formation.[19-21] These effects have been substantiated clinically in damaged hearts,[22-24] whereas no important effects were noted in normal hearts.[23-25] It has also been noted that magnesium is more likely to abolish ectopic beats caused by digitalis than those otherwise caused.[22,26] The rare paradoxic occurrence of extrasystoles

and even paroxysmal tachycardia reported following magnesium is in keeping with the occurrence of such paradoxic arrhythmias after administration of substances which ordinarily suppress them.[22,27] The effects of magnesium are generally of short duration, rarely lasting more than eight minutes.[24] Occasionally, however, the effects are more lasting. In this case, the action persisted for at least twenty minutes when it was interrupted by the administration of potassium.

EFFECTS OF POTASSIUM

During the initial period following the institution of the potassium infusion the rhythm and shape of the ventricular complexes remained unaltered, the only change noted being a slowing of the rate from 83 to 65 per minute. After twenty minutes the complexes become wider, show considerable variations in shape, and the main deflection is directed downward. The rhythm remains regular with the rate slowing further to 58 per minute (Fig. 2,*I*). Except for a brief period during which temporary reversion to auricular fibrillation occurs (Fig. 2,*J*), this picture is maintained throughout the twelve-hour period of potassium infusion. This would seem to indicate that the center of impulse formation has been depressed from the bundle to a focus below the bifurcation. However, the marked variation in the shape of the ventricular complexes also suggests a disturbance in conduction. In this connection it has been noted that conduction disturbances are, in general, worsened by potassium administration.[27]

It has been shown that digitalis, particularly in toxic doses, causes a loss of potassium from the heart muscle.[28-30] Clinically, it is well recognized that conditions associated with a loss of intracellular potassium may be associated with increased sensitivity to digitalis.[31-33] Simultaneous administration of Digoxin and potassium has resulted in fewer complications than were observed with Digoxin alone.[34] The successful use of potassium to abolish arrhythmias caused by digitalis has likewise been reported.[30,35,36] Toxic effects of potassium have been observed following its use in the presence of mild or moderate renal disease.[37]

The combination of magnesium and potassium successfully aborted the arrhythmia in this case, following which maintenance oral potassium prevented its recurrence despite subsequent redigitalization. These ions are considered to be particularly indicated in the treatment of ventricular tachycardia due to digitalis toxicity.[13,17,24,27] Potassium was administered in two other reported cases of bidirectional ventricular tachycardia, successfully aborting the paroxysm in each case.[2,27] In view of these observations it may well be that magnesium and potassium are the agents of choice in the treatment of this type of bidirectional tachycardia following digitalis administration.

SUMMARY AND CONCLUSIONS

1. A case of digitalis intoxication followed by paroxysmal tachycardia with rhythmic alternation in the direction of the ventricular complexes is reported.

2. The effects of carotid sinus pressure, intravenous magnesium sulfate injection, and potassium chloride infusion in aborting the arrhythmia are recorded electrocardiographically.

3. Electrocardiographic evidence is presented which suggests that the mechanism responsible for this arrhythmia is probably due to the action of a single focus, located in the nodal tissues above the bifurcation with alternate conduction down the right and left bundle branches.

4. It is proposed, therefore, that the simplified term bidirectional tachycardia be used to describe this form of arrhythmia.

5. It is further proposed that magnesium and potassium may be the agents of choice in the treatment of this arrhythmia following digitalis intoxication.

6. It is also suggested that, where carotid sinus pressure aborts the paroxysm, treatment with vagal-stimulating drugs may prevent recurrence of the arrhythmia.

SUMMARIO E CONCLUSIONES IN INTERLINGUA

1. Es reportate un caso de intoxication a digitalis sequite per tachycardia paroxysmal con alternation rhythmic in le direction del complexos ventricular.

2. Esseva registrate electrocardiographicamente le effectos exercite super le abortion del arrhythmia per (1) pression del sinus carotide, (2) injection intravenose de sulfato de magnesium, e (3) infusion de chlorido de kalium.

3. Es presentate datos que indica que le mechanismo responsabile pro iste arrhythmia es probabilemente initiate per un sol foco in le texitos nodal supra le bifurcation, con un conduction alternante que descende via le brancas dextere e sinistre.

4. Super le base de iste observationes nos propone que le simplificate termino "tachycardia bidirectional" es usate pro describer iste forma de arrhythmia.

5. Nos opina que magnesium e kalium es possibilemente le agentes de selection in le tractamento de iste typo de arrhythmia post intoxication a digitalis.

6. Finalmente nos mentiona le possibilitate que in casos in que un pression del sinus carotide aborta le paroxysmo, le recurrentia del arrhythmia es prevenibile per le tractamento con drogas vagostimulante.

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TRANSIENT ELECTROCARDIOGRAPHIC CHANGES SIMULATING ACUTE MYOCARDIAL INFARCTION

Interpretation of Their Significance and Report of a Case

Paul J. Sanazaro, M.D.

San Francisco, Calif.

TRANSIENT electrocardiographic changes simulating those of acute myocardial infarction have been reported as occurring during attacks of angina pectoris.[1-4] The chance recording of an unusual sequence of electrocardiographic abnormalities in a patient with atypical manifestations of coronary artery disease led to a review of these data. The conclusion was reached that the mechanism of myocardial ischemia responsible for these changes differs from that which produces "angina pectoris" in the conventional sense of the term.

CASE REPORT

L. C., a 66-year-old white man, first noted oppressive retrosternal discomfort in February, 1954. The discomfort lasted for hours or days, and was unrelated to exertion, emotion, or meals. Initially it occurred every 2 or 3 weeks, then more frequently as the pain gradually shifted into the precordium and left arm. On October 2, 1954, the chest discomfort was more severe than usual, but the patient felt well otherwise. Without premonitory symptoms he suddenly fell to the floor, unconscious and breathing noisily. He recovered within a few seconds and was alert. Pain subsided within an hour but later recurred, along with syncope, while the patient was resting in bed. He was hospitalized that day. Nitroglycerin had never relieved the pain. Dyspnea, orthopnea, and palpitation were denied.

A physical evaluation one year prior to entry, including an electrocardiogram and complete gastrointestinal study by x-ray, revealed only emphysema and essential hypertension with a blood pressure of 150/90 mm. Hg. Past history was noteworthy only for the presence of persistent dry cough, with occasional paroxysms that produced light-headedness. This had improved when smoking was discontinued.

Physical examination revealed a temperature of 98.2° F. (oral), pulse 80 and regular, respirations 20 per minute, and blood pressure 160/90 mm. Hg. The patient was a florid-faced, robust individual. The retinal arterioles were moderately narrowed. The chest was emphysematous. The area of cardiac dullness could not be determined. The heart sounds were faint; A_2 was louder than P_2, and no murmurs or thrills were noted. The left pedal pulses were not palpable. No abdominal organs or masses were felt. There was no cyanosis or clubbing of the fingers. Neurologic examination showed no abnormalities.

Results of laboratory tests were as follows: hemoglobin, 16.1 gram per cent; red blood cell count, 5.19 million per cu. mm.; white blood cell count, 11,700; differential count, normal. Uncorrected sedimentation rate was 16 mm. per hour; the packed cell volume was 50 c.c. per cent. Urinalysis was normal. The serologic test for syphilis was negative. An x-ray film of the chest revealed normal cardiac size and contour and emphysema of the lungs. An electrocardiogram taken the day of admission (October 2, 1954) showed no abnormalities (Fig. 1).

From the Department of Medicine, University of California School of Medicine, San Francisco, Calif.

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The patient remained afebrile, and there was no subsequent change in his white blood cell count or sedimentation rate. He noted occasional aching in his left chest and arm, which was unrelieved by nitroglycerin. On October 6, during a bed bath, he was observed by the nurse to

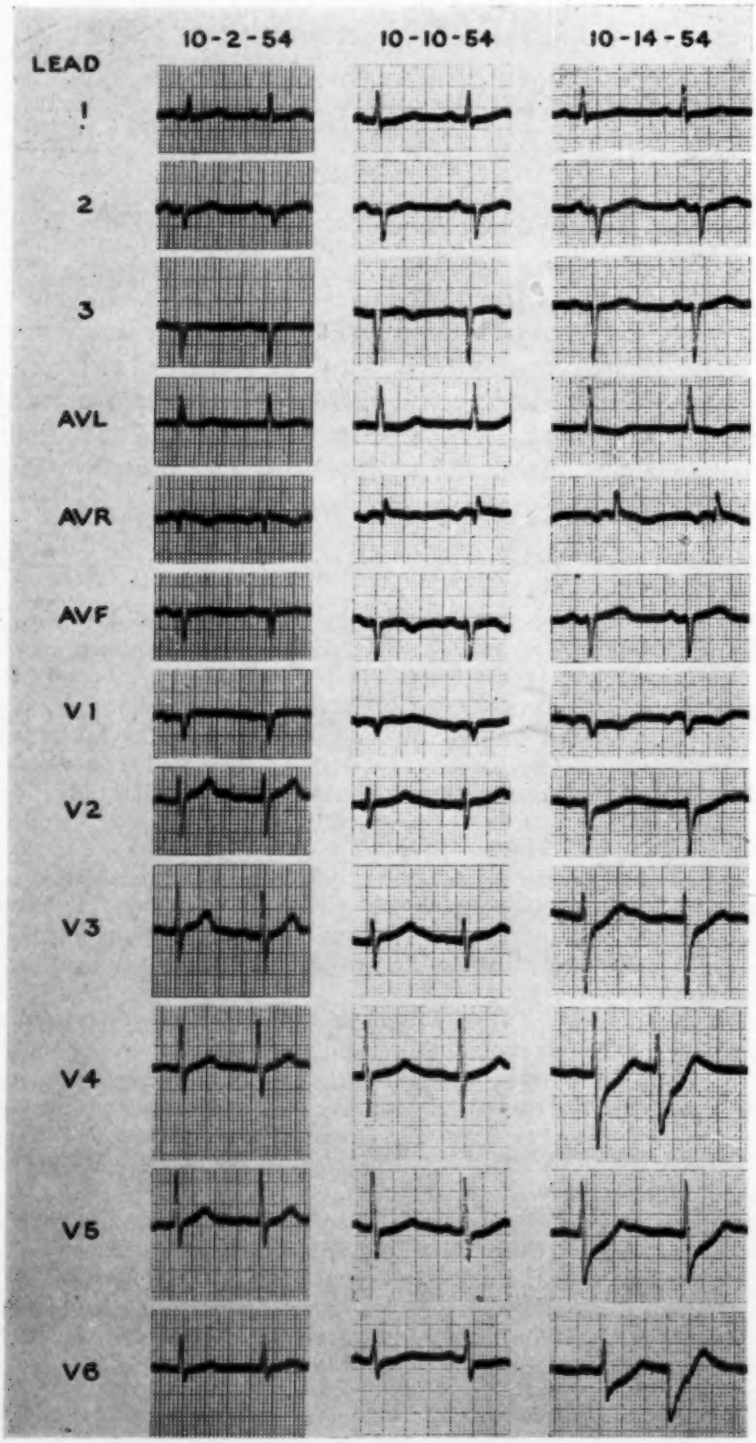


Fig. 1.—Routine electrocardiograms taken the first, eighth, and twelfth days of hospitalization. Chest pain developed as the precordial leads were recorded on October 14.

become rigid and cyanotic, with eyes and tongue protruding; the radial pulse was said to be rapid and bounding. The episode lasted only a few seconds, and the patient promptly regained consciousness. The following day he complained of sudden faintness and of a fluttering sensation in his chest. A physician listening over the apex described a rapid and completely irregular arrhythmia which stopped abruptly. This recurred later in the day and was recorded (Fig. 2). On October 9, during an attack of near syncope associated with sharp, severe chest and left-arm pain, the patient's blood pressure was 198/120 mm. Hg. A continuous electrocardiographic tracing was taken shortly after the onset, and is shown in Fig. 3. Electrocardiograms taken on October 10 revealed changes suggestive of posterior myocardial infarction, which disappeared by October 14. Pain was present while the precordial leads were being recorded on October 14. Following his discharge from the hospital on October 16, the patient had infrequent mild chest pain but no further faintness or syncope. Blood pressure has remained in the range of 140-150/90 mm. Hg. He became asymptomatic within 2 weeks.

Final diagnoses were arteriosclerotic heart disease with coronary insufficiency and Morgagni-Adams-Stokes syndrome due to unknown cardiac mechanism.

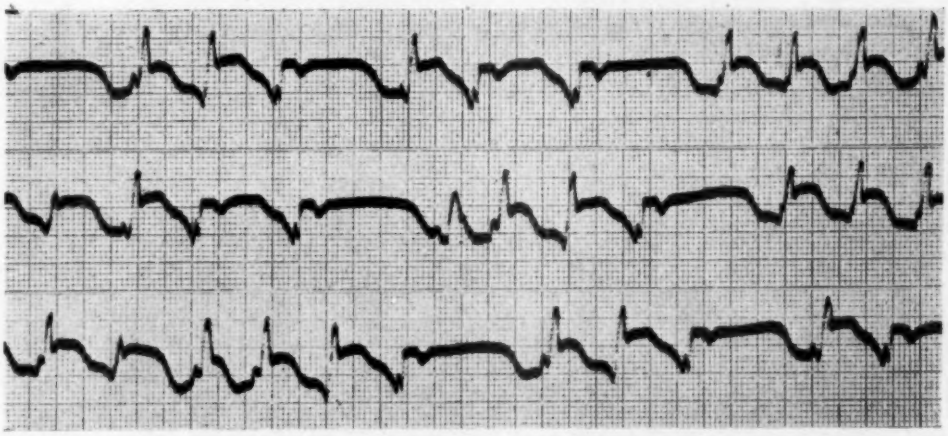
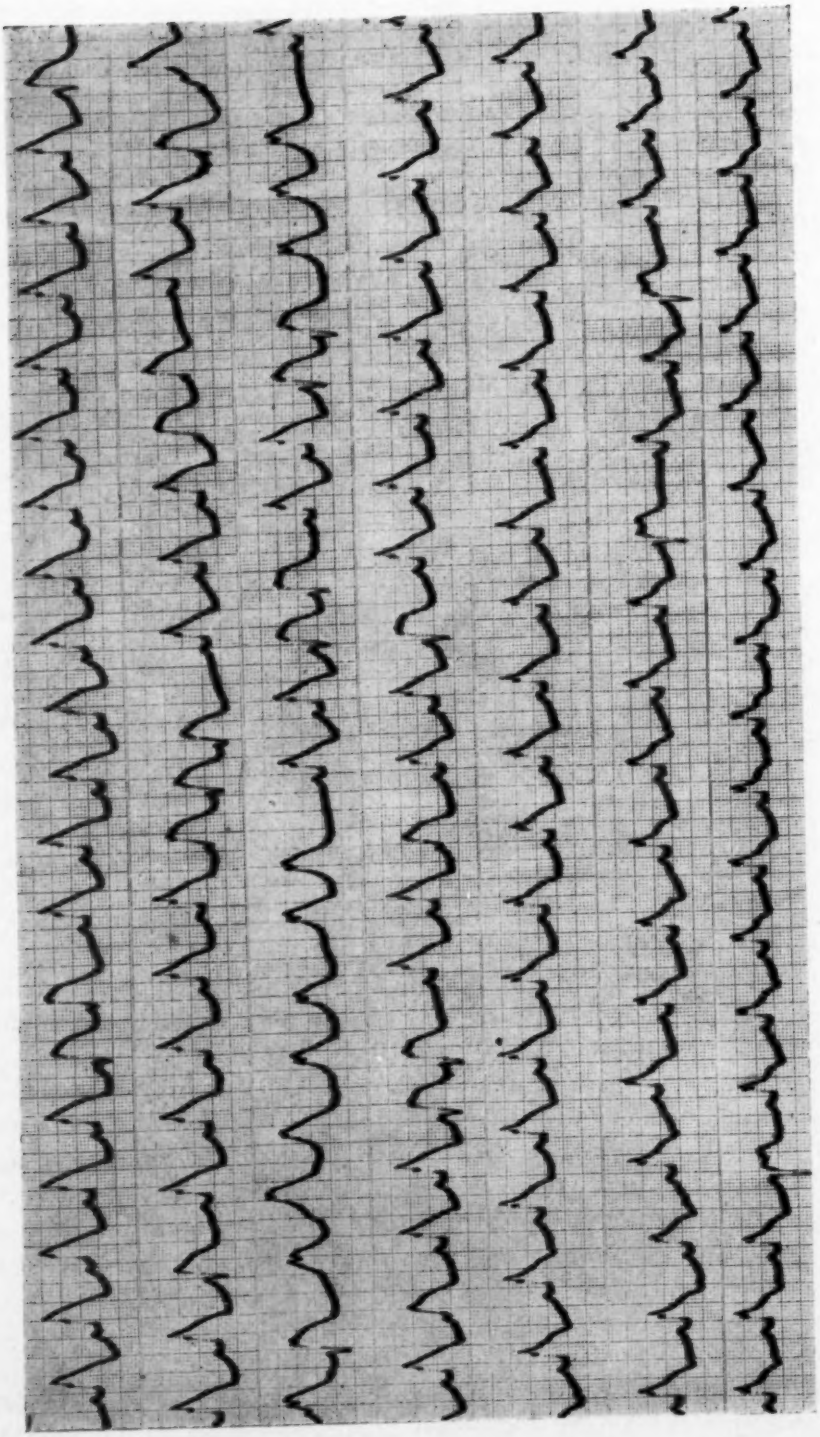


Fig. 2.—October 7, 1954. Lead II, continuous strip. Multiple premature beats of supraventricular origin displaying pattern of acute myocardial infarction.

DISCUSSION

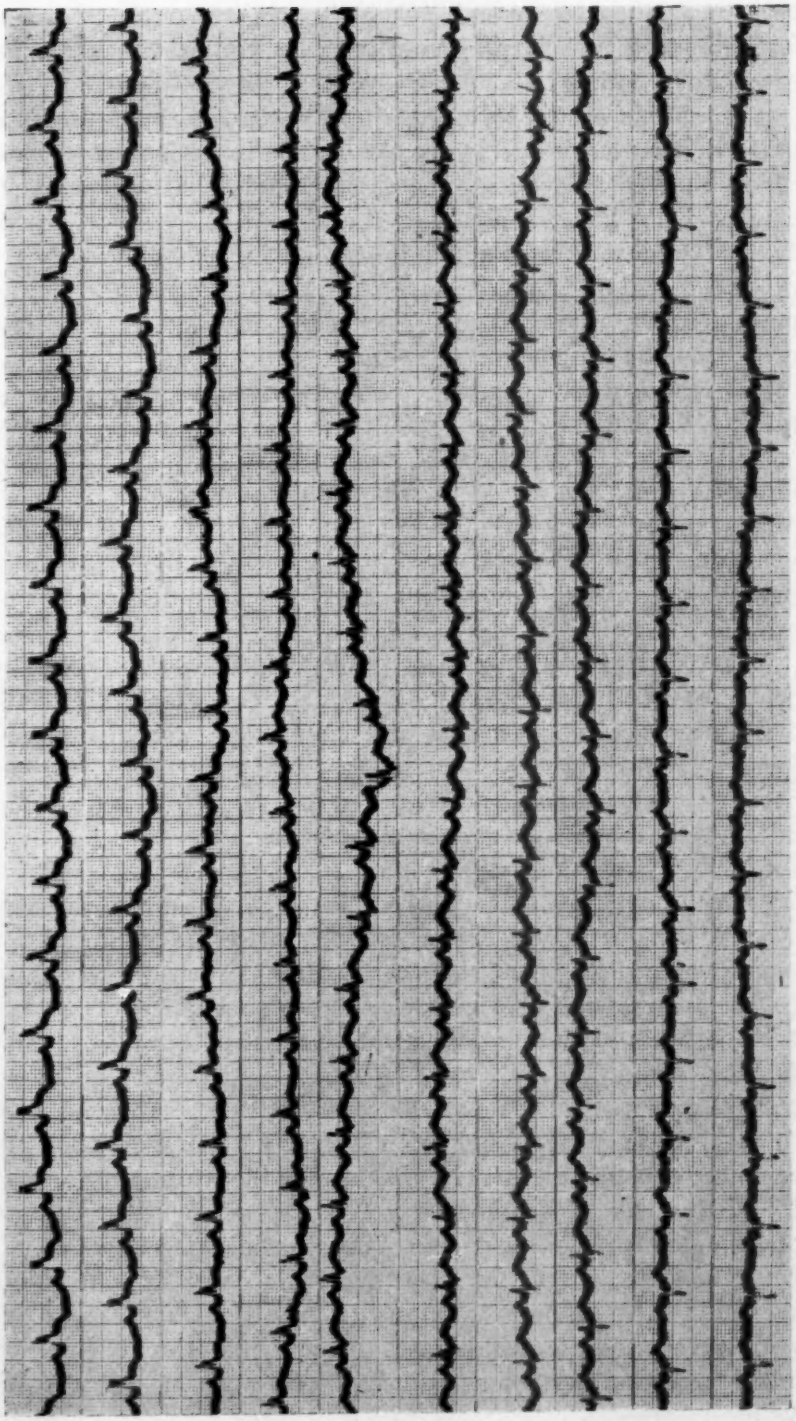
The continuous tracing illustrated in Fig. 3 demonstrates a number of unique changes. Within 8 minutes complete R-T fusion regresses serially to the contour of complete bundle branch block with concordant S-T segment elevation, followed by the gradual reappearance of the normal S with isoelectric S-T and upright T wave. So far as is known, no similar electrocardiographic changes in a human subject have been reported, although they are common in animals after experimentally induced temporary occlusion of a coronary artery.[5,6] In dogs, occlusion of the anterior coronary artery or of both main branches results in R-T fusion within ½ to 3 minutes, with return to the normal pattern within 3 minutes following release of the occlusion. In repeated experiments on the same animal, resultant changes appear more rapidly and are of longer duration as the degree and extent of accumulated myocardial damage increase.

It is noteworthy that such acute experiments in animals do not reproduce the characteristic S-T segment depression that is observed during spontaneous or induced attacks of angina pectoris and is accepted as reliable evidence of transient myocardial ischemia. In dogs, temporary interference with coronary blood flow only inconstantly produces initial variable T-wave inversion, but



A.

Fig. 3,*A*.—(For legend see opposite page.)



B.

Fig. 3.—*A* and *B*. October 9, 1954, Lead II, continuous strip. Sinus rhythm, bundle branch block with concordant S-T segment elevation, interrupted by idioventricular rhythm and septal premature beats having configuration of acute myocardial infarction. The patient was experiencing near syncope, chest pain, and palpitation during the period of the tracing.

the subsequent changes are those of acute myocardial infarction, even though actual tissue necrosis does not occur.[7] The specific physiologic basis of the S-T segment depression ascribed to subendocardial ischemia is actually unknown, since it cannot be reproduced experimentally under conditions which induce transient myocardial ischemia.[8]

Review of a number of case reports describing electrocardiographic changes similar to those of myocardial infarction but occurring in patients with "angina pectoris" revealed that in every instance the manifestations of angina were atypical.[1-4] The patients had noted either the abrupt onset of frequent and disabling episodes of anginal pain or a definite change in their previously typical symptom complex, the pain occurring usually at rest, lasting many minutes or hours, often unrelieved by nitroglycerin. The stated criterion for the diagnosis "angina pectoris" was the absence of subsequent clinical, electrocardiographic, or laboratory signs of myocardial necrosis. The described manifestations would be more suitably classified as coronary failure,[9] coronary occlusion without infarction, or the premonitory syndrome of myocardial infarction. It is reasonable to assume that during the period in which the electrocardiographic observations were made, the coronary circulation was temporarily unstable, resulting in transient severe impairment of blood supply to a given area of the myocardium. Although restricted, the blood supply was nevertheless sufficient to prevent ischemic myocardial necrosis.

Transient, bizarre QRS abnormalities, among which were marked degrees of intraventricular block, together with S-T segment elevation to the point of R-T fusion, have been recorded during the course of intravenous angiocardiography in children with congenital heart disease and cyanosis.[10] Circulatory collapse, pulmonary edema, coronary artery spasm, and toxic effect directly in the myocardium were the postulated causes of these changes.

Of interest in Fig. 2 are the frequent runs of premature beats, presumably septal in origin, which have the typical appearance of acute posterior myocardial infarction (Lead II). The "unmasking" of otherwise occult infarction by premature beats, while rare, is considered a reliable observation.[11] In this instance there were no enduring electrocardiographic changes of infarction, although premature beats of similar configuration recurred two days later (Fig. 3), along with short runs of idioventricular rhythm.

Also worthy of comment is the occurrence in Fig. 3 of concordant S-T segment elevation, with the major QRS deflection of the bundle branch block pattern. This exception to the general rule that bundle branch block obscures or prevents the appearance of the usual S-T and T-wave changes of myocardial infarction has generally been noted early in the course of myocardial infarction.[12]

The episodes of syncope and faintness experienced by the patient in this report are unexplained. No periods of auriculoventricular block or cardiac standstill were observed, and Fig. 2 illustrates the only arrhythmia demonstrated. During the periods of faintness the patient's blood pressure was significantly elevated above his basal level.

SUMMARY

A series of transient electrocardiographic changes are reported which simulate those obtained during experimental temporary coronary artery occlusion

in animals. The patient in whom they were recorded was experiencing episodes of syncope and atypical anginal pain and displayed no signs of myocardial infarction. Similar fleeting electrocardiographic abnormalities suggesting myocardial infarction have been reported in the literature as occurring in angina pectoris. A review of the published data indicates that a more serious disturbance of the coronary circulation is responsible, best described as the syndrome intermediate in degree between transient myocardial ischemia responsible for angina pectoris and coronary occlusion producing myocardial infarction.

SUMMARIO IN INTERLINGUA

Es reportate un serie de transiente alterationes electrocardiographic que simula le alterationes obtenite durante le temporari occlusion experimental del arteria coronari in animales. Le patientes in qui illos esseva registrate experientiava a ille tempore episodios syncopal e atypic dolores de angina e non exhibiva ulle signo de infarcimento myocardiac. Simile anormalitates electrocardiographic de character transiente que simulava infarcimento myocardiac es a trovar in le litteratura como occurrentias in casos de angina de pectore. Un revista del datos publicate indica que un plus serie disturbation del circulation coronari es responsabile pro iste phenomeno. Il se tracta de un disturbation que es le melio describite como un syndrome intermediari in grado de severitate inter le transiente ischemia myocardial que es responsabile pro angina de pectore e le occlusion coronari que produce infarcimento myocardiac.

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COMPLETE HEART BLOCK WITH RHYTHMICALLY RECURRING DECAY AND FAILURE OF THE IDIOVENTRICULAR PACEMAKER

J. H. PALMER, M.D., F.R.C.P.

MONTREAL, CANADA

THE following case history concerns a patient with complete heart block in whom Stokes-Adams attacks occurred regularly over a period of several days as a result of rhythmically periodic slowing and failure of the idioventricular pacemaker. It appears to be clinically unique, and suggests certain conclusions of importance to the subject of ectopic rhythms. An attempt will be made to show that the peculiar qualities of acceleration and deceleration displayed by the ventricular rhythm present in this case are related to those of certain paroxysmal ectopic tachycardias and perhaps to nonphasic sinus arrhythmia.

CASE REPORT

H.B., a male machine-shop worker, aged 63 years, was admitted to the Montreal Neurological Institute, and later to the Royal Victoria Hospital, on Jan. 14, 1947.

He gave a history of having had, once or twice a month during the past three years, episodes of transient loss of awareness. Occasionally he would fall, but there was never any convulsion. He had mild shortness of breath on exertion, but no other abnormal cardiovascular symptoms.

Four days before admission he slipped on some ice and fell, striking his head. He was momentarily stunned but was able to walk home. Later that day he abruptly became unconscious and fell, this time loosening some teeth. Similar attacks then occurred frequently. He was sent from his home in an outlying town to the hospital with the tentative diagnosis of brain injury.

Physical examination revealed no gross abnormalities apart from those of cardiac rhythm, to be described later. The temperature was 100.6° F. and the blood pressure was 190/60 mm. Hg. X-ray pictures of the skull and chest were normal.

From the time of admission, classical Stokes-Adams seizures occurred regularly at intervals of less than two minutes, unless they were controlled by epinephrine. Ten to fourteen seconds after the beginning of ventricular arrest the pupils would dilate, the respiratory rate would increase, and obvious loss of consciousness would occur, with twitching of the facial muscles, adversion of the head and eyes to right or left, and occasional convulsive movements of the limbs. The duration of ventricular arrest was just over thirty seconds in almost every case, although one period was timed at forty-one seconds. Ventricular activity lasted for less than one minute; the rate during the first half of this period, after increasing slightly at first, became fairly constant at 43-44, and then gradually slowed to 30. This general pattern remained unchanged during the next four days.

Lumbar puncture was done, six attacks occurring while the needle was in situ. The pressure, which varied between 160 and 450 mm. of water, was considered to be fundamentally normal, with the rise during an attack not having time to subside before another occurred.

From the Department of Medicine, McGill University, and the Royal Victoria Hospital, Montreal.
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Subcutaneous injection of 0.5 c.c. of 1:1,000 Adrenalin solution brought about cessation of the attacks at once. They began to recur in approximately ninety minutes and, to control them, it was necessary to repeat the Adrenalin injection every one and one-half to two hours during the next four days. Periodically during this time, for teaching purposes and to investigate the effect of other drugs, the Adrenalin was withheld. Ephedrine, 45 mg. four times daily by mouth, not only did not control the attacks but failed to make any appreciable difference in the amount of Adrenalin necessary. Adrenalin in oil in 2 mg. dosage subcutaneously was ineffective. Subcutaneous Neosynephrine controlled the attacks, but was considered inferior to Adrenalin. Nitroglycerin did not influence the course of events. Atropine sulfate, 0.6 mg. intravenously, caused no obvious effect and neither did 1.2 mg. subcutaneously on another occasion.

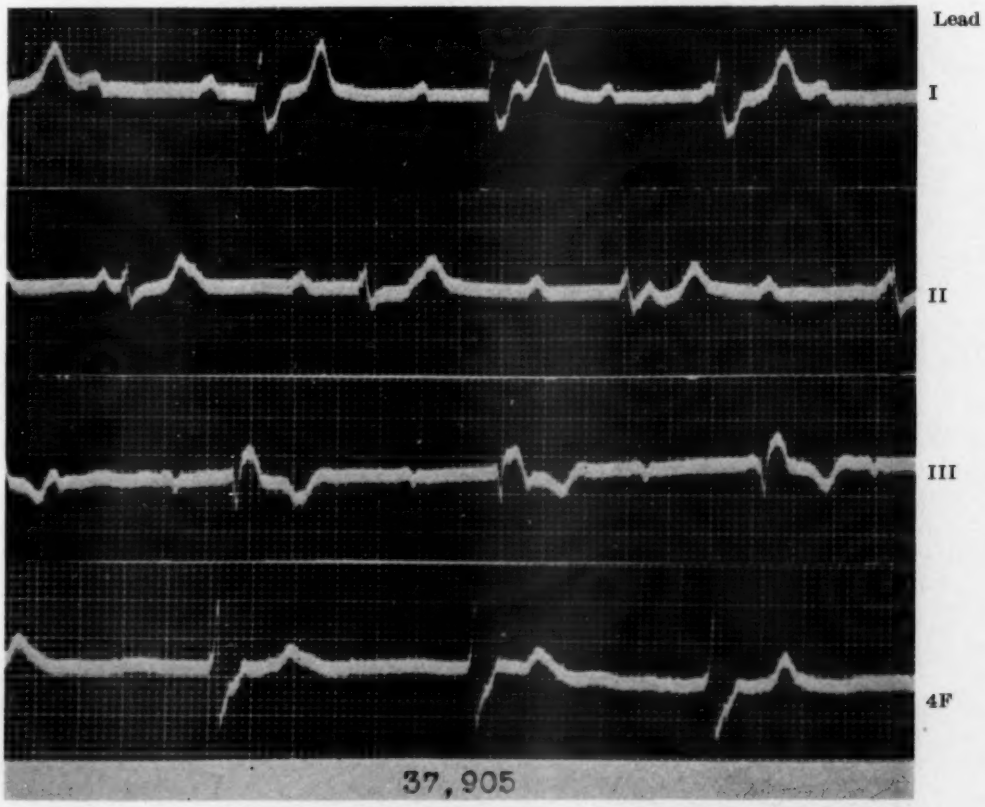

Fig. 1.—Electrocardiogram taken on Jan. 15, 1947, during period of Adrenalin control.

An electrocardiogram was first made on January 15, during a period of Adrenalin control, and showed complete heart block with an auricular rate of 110 and a ventricular rate of 45. The QRS complexes were of a form which, in a patient with normal auriculoventricular conduction, would have been considered characteristic of right bundle branch block (Fig. 1). Several tracings were made on the same day while attacks were occurring (as a result of withholding Adrenalin), and all showed the same general pattern, namely, complete heart block with rhythmically recurring periods of ventricular asystole (Fig. 2). The auricular rate was fastest (115 per minute) at the end of ventricular activity, and slowest (90 per minute) at the end of the asystolic period. The periods of ventricular activity, which lasted for about forty seconds, began with the rate accelerating slightly for three or four beats and then remaining constant during the next twenty seconds at 43 per minute. Gradual slowing now occurred until four to five seconds separated the final two beats. Aberrant ventricular complexes were sometimes seen at the beginning or end of periods of activity.

On January 17, simultaneous electroencephalograms and electrocardiograms were made, and several successive seizures were observed. No significant abnormalities were observed during ventricular activity. About ten to fifteen seconds after its cessation there was a gradual slowing of brain waves, followed by loss of consciousness, some movement of the head and extremities, and gasping. During this time, the electroencephalogram was obscured by movement artifact but, following return of ventricular activity, slow waves were usually observed lasting for a few seconds, after which the electroencephalogram returned to normal.

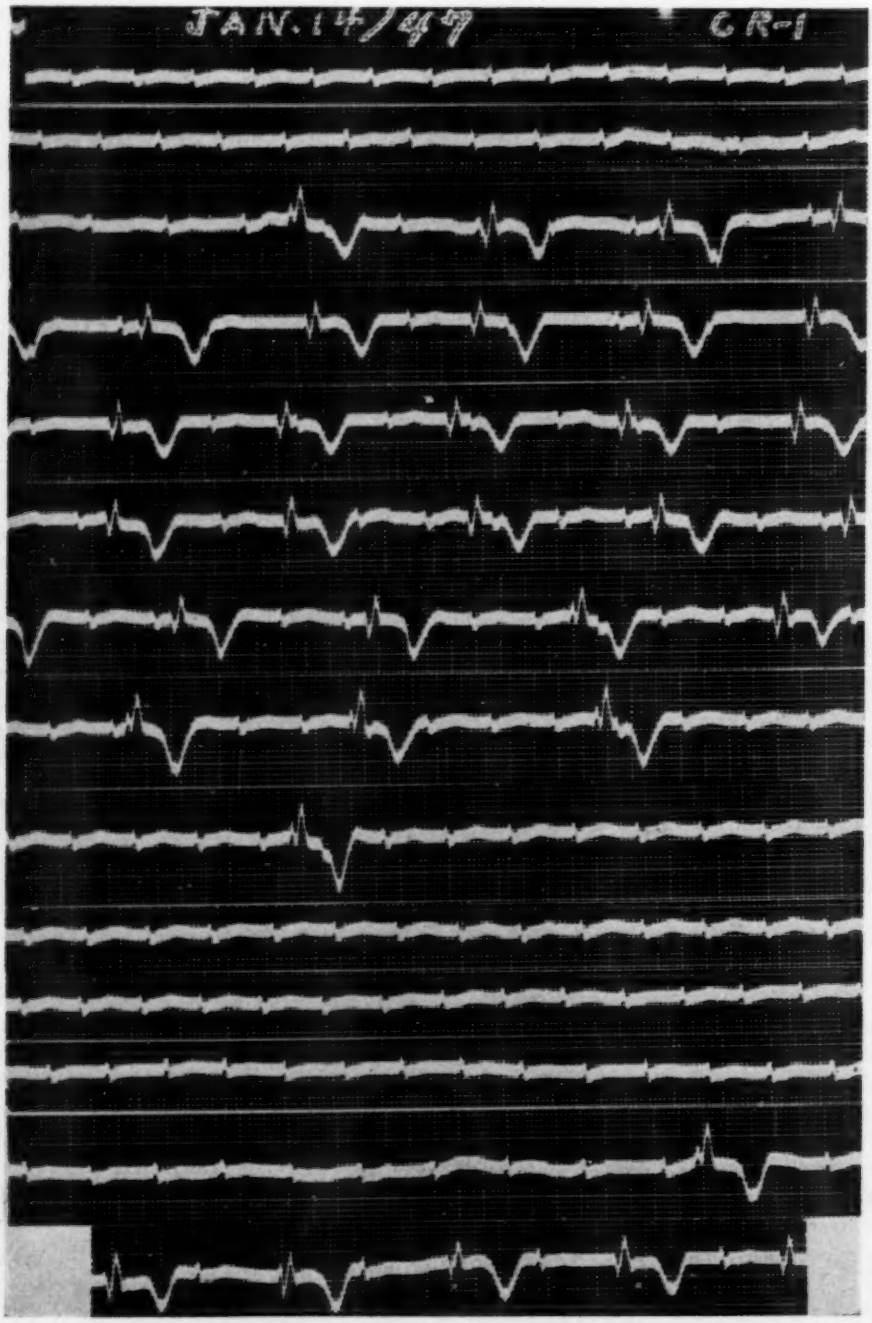

Fig. 2.—Electrocardiogram taken on Jan. 15, 1947, to show general pattern of attacks. Lead CR_1, continuous tracing.

Other laboratory investigations included the following: Wassermann reaction, negative; arm-to-tongue circulation time (Decholin), eleven seconds; venous pressure, 100 mm. of water, rising during attacks to 160 to 180. The urine showed a trace of albumin and some granular casts on admission, but later was normal. The white cell count was 11,200 on January 19, and 9,500 on January 21. The uncorrected sedimentation rate was 30 mm. on January 21 and 46 mm. on January 30. On January 17 the blood nonprotein nitrogen level was 34.5 mg. per 100 c.c., and the serum sodium and potassium, respectively, 148.7 and 5.85 meq./1.

The blood pressure was 190/60 mm. Hg on admission. Thereafter, wide ranges were recorded from 120/80 to 230/110 mm. Hg. It was considered that during treatment the frequent injections of Adrenalin, which resulted in headache, sweating, pallor, and "jitteriness," contributed largely toward causing the higher readings. After normal rhythm returned on January 18, the blood pressure remained at about 145/90 mm. Hg.

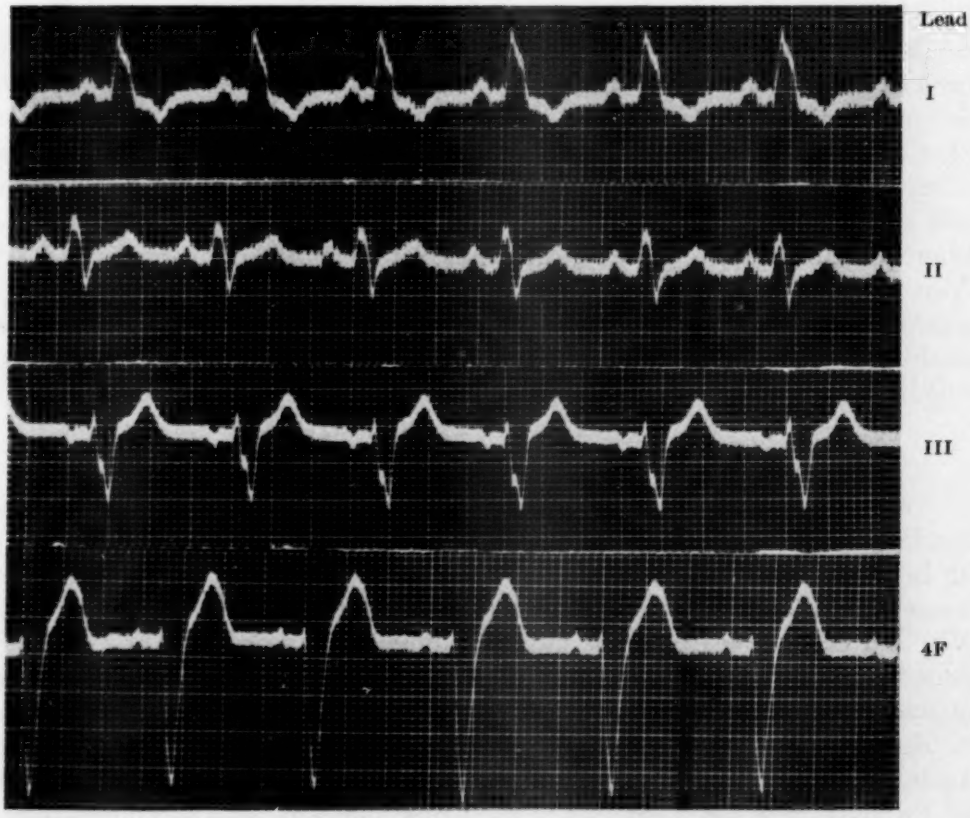

Fig. 3.—Electrocardiogram taken on Jan. 18, 1947, after reversion to normal rhythm.

On the afternoon of January 17, the usual dosage of Adrenalin was effective for from three to four hours. Early in the morning of January 18 (that is, four days after onset) the patient had his last injection, and soon afterward it was noted that his pulse rate had increased to about 90 per minute and was regular. An electrocardiogram (Fig. 3) showed normal rhythm with left bundle branch block and a normal P-R interval. This persisted unchanged until he left the hospital on February 5.

Following his return home, he remained quiet for a few months and then resumed light mechanical bench work for three years, until he was superannuated. He is still active and does a considerable amount of walking. During the seven years that have elapsed he has had three attacks in which he fell, and a few others of less severity. He continues to have left bundle branch block with a normal P-R interval (September, 1954), the electrocardiogram remaining similar to that shown in Fig. 3.

COMMENT

It seems unlikely that complete block had been present for long before the patient's arrival at the hospital, as his local physician had not remarked on slowness of the pulse, which, at a maximum rate of 43, undoubtedly would have attracted his attention. There was slight fever for one day only and slight leukocytosis, and it was considered possible that there had been a small, silent cardiac infarction involving the septum, the only localizing sign being the heart block itself. This conception was adopted as a working basis in the management of the case.

The history of transient blackouts for three years suggests that conduction was defective during that period and that the left bundle branch block found when the rhythm became normal, and which has persisted since then, may have been already of fairly long standing. Whether this is so or whether it appeared as part of the recent acute incident is immaterial. In either event, it seems likely that the complete block was due to bilateral branch block rather than to a lesion of the main bundle. Supporting this argument is the finding that the new pacemaker was in the left ventricle rather than high in the right bundle branch, the most likely place for it to appear if the main bundle had been blocked. Also, it has been shown by Stokes[1] that whenever complete block develops in a patient who already has a bundle branch lesion, there is a greatly preponderant likelihood, if the P-R interval until then has been normal, that block of the other bundle branch has taken place.

DISCUSSION

An outstanding feature of ectopic rhythms and tachycardias is their remarkable constancy of rate. While there may be some variation from beat to beat in ventricular tachycardias[2] and in the ventricular rhythm of complete heart block, it is not great. In several cases of supraventricular tachycardia, Feil and Gilder[3] measured the maximal differences in the lengths of cycles and found them rarely to exceed 0.01 of a second. Occasionally, in long-continued paroxysms, there may be slight variations in rate from hour to hour or from day to day. Our concern is not with these, but rather with bouts of paroxysmal tachycardia in which fairly abrupt and readily observable waxing and waning of the pulse rate have occurred. Such cases are rare and, whenever their crescendo-decrescendo nature has been pronounced, there has been also a tendency to rhythmical repetition of paroxysms. Thus, Parkinson and Mathias,[4] in 1915, described a case of supraventricular tachycardia in which paroxysms, separated by a few normal beats, were repeated every two to three minutes for four days; in each, the rate gradually increased from 140 to 250 per minute and then quickly fell to its original rate before terminating. I have records of two similar cases (unpublished material), in one of which the regularity of recurrence and the crescendo-decrescendo character of the bouts are represented in Fig. 4. This sequence persisted for four days; then, under the influence of digitalis, there appeared shorter paroxysms of a few seconds' duration which recurred rhythmically at intervals of a few seconds and which, on auscultation alone, could have

been mistaken for nonphasic sinus arrhythmia. A case reported by Paul White[5] seems to have shown similar short paroxysms rhythmically repeated.

The resemblance of these bouts of ectopic tachycardia to those of the rythmically recurring periods of ventricular activity seen in the case of heart block now being reported is obvious. Both are ectopic rhythms and in both the impression is given that there has been a regularly recurring decay and eventual failure of the pacemaker. It is not the intention of this communication to discuss the mechanism whereby this is brought about. That it can occur, however, in the ventricle, where the vagus has no direct action, is important in that it makes it likely that the vagus need not be concerned in the supraventricular cases.

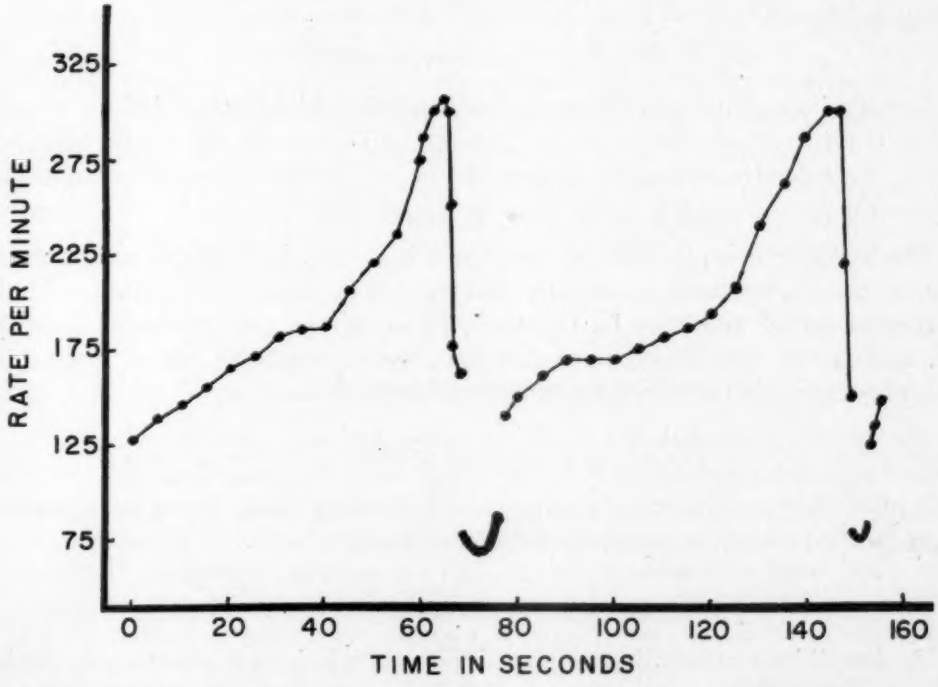

Fig. 4.—Graph showing fluctuations of rate in author's case of paroxysmal auricular tachycardia. Beaded line represents ectopic rhythm; solid line represents normal rhythm. (See text.)

Electrocardiographic tracings which have recorded the onset or offset of individual paroxysms of tachycardia have often[6] shown that there is a tendency for the ectopic pacemaker to gain slightly in speed after it starts. Again the termination of some paroxysms, whether spontaneous or induced by drugs or carotid sinus pressure, is not uncommonly marked by a decrease in rate involving the last two or three beats; two examples of this are illustrated by Parkinson and Papp.[7] It seems reasonable to conclude that these isolated instances of waxing or waning should not be differentiated except in degree from the others already referred to.

A consideration of so-called nonphasic sinus arrhythmia now becomes pertinent and suggests the possibility that the sinus node itself at times may show rhythmic phenomena indistinguishable from those of the ectopic foci. This

arrhythmia was first described satisfactorily by de Meyer[8] as "acceleration sinusale intermittante." While it was not found in healthy patients, it was considered by him to be fairly common and he was able to observe in each patient long periods of regularly alternating waxing and waning of rate which were entirely independent of respiration. Because they were not suppressed by carotid sinus pressure or by physostigmine, he concluded that they were not the result of vagus activity. This argument, however, is somewhat weakened but not ruled out by the finding that they can be abolished by atropine. Others have agreed that this arrhythmia is usually associated with abnormal conditions; it is found particularly in older people with heart disease,[9] in young subjects after exercise and during convalescence from febrile illnesses,[10] and sometimes during digitalis therapy.[11]

SUMMARY AND CONCLUSIONS

A case of complete heart block is described, in which Stokes-Adams seizures resulted from rhythmically recurring slowing and arrest of the ventricular pacemaker. This persisted for several days, during which time it could be suppressed temporarily by Adrenalin. The case is apparently unique in the literature.

The hypothesis is put forward that there is in ectopic rhythms, under certain circumstances, a tendency to waxing and waning of rate. Occasionally this becomes pronounced and may be rhythmically recurrent. It probably is pathologic, and is not dependent on vagus activity. Nonphasic sinus arrhythmia may be an expression of the same tendency in the sinus node.

The phenomenon has not been the subject of any considerable study. It is important clinically in that it is concerned with the beginning and the termination of ectopic tachycardias; during complete heart block it can be responsible for a hitherto undescribed variety of Stokes-Adams attack.

SUMMARIO IN INTERLINGUA

Es describite un caso de complete bloco cardiac in que attaccos de Stokes-Adams resultava ab recurrentias rhythmic de retardation e arresto del pacemaker ventricular. Isto persisteva durante plure dies. Su suppression temporari esseva possibile per medio de adrenalina. Il pare que iste caso es unic in le litteratura.

Es presentate le hypothese que in rhythmos ectopic il ha sub certe conditiones un tendentia del frequentia a accrescer e decrescer. A vices iste tendentia deveni pronunciate e pote recurrer rhythmicamente. Le phenomeno es probabilemente pathologic e non depende del activitate del vago. Nonphasic arrhythmia sinusal pote esser un expression del mesme tendentia in le nodo sinusal.

Le phenomeno ha non ancora essite subjicite a ulle studio detaliate. Illo es clinicamente importante in tanto que illo es concernite con le comenciamento e le termination de tachycardias ectopic. In complete bloco cardiac illo pote devenir responsabile pro un non ancora describite varietate de attacco de Stokes-Adams.

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Announcements

POSTGRADUATE COURSE IN BASIC ELECTROCARDIOGRAPHIC ANALYSIS will be conducted by Robert H. Bayley, M.D., and L. L. Conrad, M.D., March 5 through March 9, 1956, at the University of Oklahoma School of Medicine, 801 N.E. 13th St., Oklahoma City, Okla. Fee: $75.00.

THE UNIVERSITY OF MINNESOTA announces a continuation course in Recent Advances in Internal Medicine for Internists which will be held at the Center for Continuation Study from Feb. 13 to 15, 1956. This year's program will deal principally with recent advances in the fields of endocrinology and metabolism, renal disease, and cardiology.

Guest speaker will be Dr. Joseph W. Jailer, Associate Professor, Department of Medicine, College of Physicians and Surgeons, Columbia University, New York, N.Y., who will also give the Minnesota Pathological Society Lecture on Tuesday evening, February 14. The course will be presented under the direction of Dr. C. J. Watson, Professor and Head, Department of Medicine. Lodging and meal accommodations are available at the Center for Continuation Study.

The ninth annual meeting of the WESTERN SOCIETY FOR CLINICAL RESEARCH will be held Jan. 27 and 28, 1956, at Carmel-by-the-Sea, Calif.

Information regarding the meeting may be obtained from Arthur J. Seaman, M.D., Secretary-Treasurer, University of Oregon Medical School, Portland 1, Ore.

Book Reviews

THE BODY FLUIDS (BASIS PHYSIOLOGY AND PRACTICAL THERAPEUTICS). By J. R. Elkinton, M.D., and T. S. Danowski, M.D., Baltimore, 1955, Williams & Wilkins Company, 626 pages.

In the physiological section of the book the authors have done an excellent job in presenting the difficult and complex problems of fluid transfer in normal and diseased states. They have gone into great detail to explain how disturbances in fluid balance arise in a number of conditions, among them, congestive heart failure, cirrhosis of the liver, renal failure, diabetic ketosis, and the endocrine conditions in which fluid and electrolyte balance are disturbed. There is also an excellent chapter on body fluid problems in surgical patients. Needless to say, the authors could not discuss body fluids without describing the rôle of the electrolytes, acid-base balance, and hormones. It would be impossible to discuss body fluids without including some physical chemistry and mathematics. However, the authors have, by the use of well chosen illustrations and fairly simple mathematics, made it possible for the average physician to read this book without difficulty.

The therapeutic section bears a direct relationship to the physiological section which precedes it. The physician will not find in it a ready routine to follow blindly. What information he will get out of this section will depend to a large extent on how well he has mastered the material in the sections concerned with basic and applied physiology. This book, then, is really a textbook and should be studied carefully from cover to cover. It cannot be used as a general reference book except by those who have done special work in this field.

The authors have gone to great pains to have this book up-to-date in a field where knowledge is rapidly expanding. Each chapter is followed by a good bibliography.

A.C.D.

THE BIOLOGIC EFFECTS OF TOBACCO. Edited by Ernest L. Wynder, Boston and Toronto, 1955, Little, Brown & Company, 215 pages.

Dr. Wynder, who is well known for his studies on the relationship of tobacco smoking to lung cancer, has tried, with the help of seven collaborators, to present the scientific facts on the extremely controversial subject of the effects of tobacco on man. An excellent chapter on chemistry written by Alvin Kosak is followed by a careful review of the pharmacology by Charles J. Kensler. The subjects of allergy, the gastro-intestinal tract, and neoplastic diseases each form a separate chapter. The chapter on the cardiovascular system is written by Ellen McDevitt and Irving Wright. As far as the cardiovascular system is concerned nicotine is the only substance in tobacco which has been proved to produce a deleterious effect in susceptible individuals. Although there is no evidence that tobacco is an etiologic factor in peripheral arteriosclerosis, coronary artery disease, Raynaud's syndrome, cerebral vascular disease, or hypertension, its continued use by individuals suffering from these conditions can accentuate the symptoms and unfavorably influence the clinical course. This is particularly true of thromboangiitis obliterans, where, unless tobacco is discontinued, the patient will usually suffer amputation.

Nicotine, or smoking tobacco, has been shown to cause changes in the ballistocardiogram, electrocardiogram, skin temperature, and blood flow. These changes appear to be reversible in the normal individual. There does not seem to be any support for the widespread belief that alcohol will counteract the deleterious effects of nicotine.

Drs. McDevitt and Wright are of the opinion, after a careful review of existing data, that "although the average longevity appears to be unfavorably influenced by the continued use of tobacco, further careful, statistical study of many additional factors is indicated in this area."

The bibliography at the end of each chapter furnishes excellent reference material. There is also a good subject index.

A.C.D.